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PHYSIOLOGY OF THE VOCAL CORDS IN PHONATION AND RESPIRATION

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The year 1886 saw a splendid and noteworthy meeting of the American Laryngological Society. Many of the papers read were of theoretic rather than practical importance, which for that era was, perhaps, a little unusual. The president, Harrison Allen,[1] of Philadelphia, evidently considered himself duty bound to lend the support of his office to such a deviation and said in his presidential address:

> . . . I hear without sympathy the expression of opinion that a paper is good in the degree that it is practical. Can we not with propriety look forward to the time when every Fellow will be identified with papers based upon results gained by experiments upon the lower animals, or upon morphological data, as well as those secured from the operating chair? We will be raised in our own esteem in the degree that we add a little more science to our art.

By no means least among these addresses stressing science rather than the art was an amazingly well illustrated paper entitled "The Laryngeal Image Photographed During the Production of Tones in the Singing Voice," by Dr. Thomas French,[2] of Brooklyn. The high lights of this address, which was illustrated by photographs of the larynx, represent the fundamental basis of much of the modern concept of the physiology of the true vocal cords. The method used for taking the photographs had been described previously.[3] These photographs even if presented today as modern contributions, would, despite their age, excite the admiration of any audience and compare favorably with the best of modern efforts. Some of them are reproduced from *The New York Medical Journal* (fig. 1). It is interesting to note that the author in his original method, described in 1884, used the sun as a source of light. In a later method, described in 1897,[4] a 2,000 candle power arc lamp was utilized. As a direct result of the study of these photographs,

1. Allen, H.: Presidential Address, Tr. Am. Laryng. A. **8**:4, 1886.
2. French, T. R.: The Laryngeal Image Photographed During the Production of Tones in the Singing Voice, Tr. Am. Laryng. A. **8**:107, 1886.
3. French, T. R.: On a Perfected Method of Photographing the Larynx, New York M. J. **40**:653, 1884.
4. French, T. R.: Laryngeal and Postnasal Photography with the Aid of the Arc Light, New York M. J. **65**:105, 1897.

French was able to describe certain important physiologic phenomena. Contributions on this subject by many laryngologists during the succeeding fifty and more years have served to establish only more firmly the accuracy of French's observations. From them one can subtract nothing as having been disproved in the course of time; to them one can add only a little, even after five decades. As the basis of my paper, then, I quote from this article by French,[2] and shall add those embellishments required to bring his observations up to date.

Different individuals often employ different mechanisms in the production of the voice, and therefore the appearances of the larynx under similar circumstances are often very different. In studying the anatomy of the larynx, we find that, in the normal state, there are always certain structures, but do we ever find the

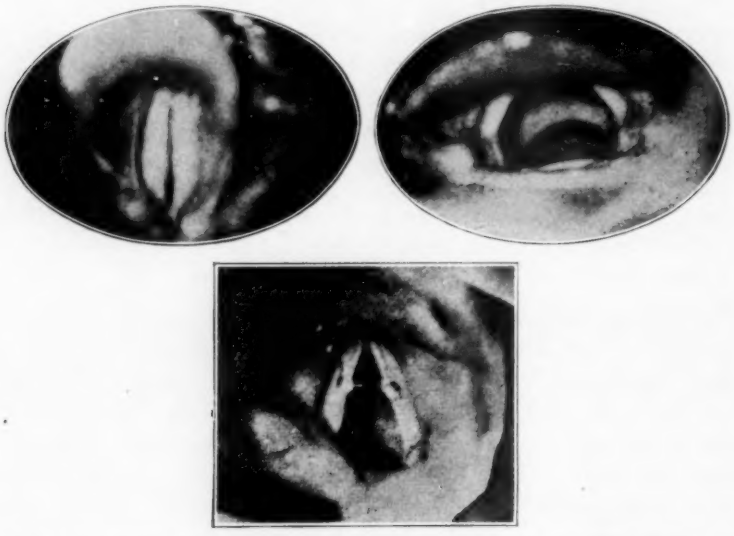

Fig. 1.—Photographs of the living human larynx by Thomas French, M.D., 1884 (reprinted from *The New York Medical Journal*[3]).

shape and groupings of these structures in different subjects exactly the same? I believe not, any more than we find the shape and groupings of the features of any two faces exactly alike. The minute differences in the size, shape and relation of the various structures give rise to some difference in the movements of the parts, and therefore it will be found that no two larynges act exactly alike under similar circumstances. Suppose we compare, with the aid of the laryngoscope, the larynges of two subjects having the same class of voice. In the first place, we find that the relative positions of the parts vary somewhat, and therefore the shapes of the larynges as a whole. We also find that the movements of the various structures in the acts of respiration and phonation differ somewhat. Suppose now that we place a mirror in each of the throats, and examine them at the same time while they are singing the same tone. We find that there is some difference in the positions of the vocal bands. The difference may be marked, or it may be so little that it will be hard to see. But there will certainly be a difference.

It is taught by many writers that certain changes always take place in the positions of the vocal bands at certain notes in the scale as the voice ascends, some asserting that these changes occur suddenly. That certain changes do occur in many larynges while singing up the scale can be easily demonstrated, and some of these will be shown here to-day; but these changes are, I believe, almost always gradual, not sudden. In several subjects whose voices have not been trained, I have detected a slight sudden change in tension of the vocal bands at certain notes, where a break in the voice could be distinctly heard. Though I have looked for it in other untrained voices under similar conditions, a sudden change, if it occurred, was not apparent in the laryngoscopic mirror. In most larynges, whatever the peculiarity in the shape of the glottis, in the production of lower tones of the voice the chink is open widest in its posterior part. In the production of the middle tones, the chink is usually linear in shape; and in the high tones the chink is open widest in front, and the glottis may or may not be closed behind. In contralto and bass voices, however, the glottic chink may not become linear even in the highest tones. Both by reflected and by transmitted light the vocal bands are seen to be thick and wide in the production of the lower tones, and to grow gradually thinner and narrower as the voice inclines upward. In all larynges the vocal bands are shortest in the production of the lower tones, and longest in the middle tones, and grow [functionally] shorter again in the upper tones. . . . I will not at this time express an opinion regarding the registers of the human voice. The study of the voice in this new light has just begun, and a large number of observations must be made before definite conclusions can be reached. The point which I desire particularly to make clear in this paper is that, while it is probable that there are certain laws which apply in all cases to the parts of the vocal bands which must vibrate for the production of certain tones of the voice, no single description of the laryngoscopical appearances which are present in the production of tones in any part of either of the classes of voice will suffice to cover all cases. Larynges differ so much in size and shape as to make it impossible for the same conditions to appear in all. The student becomes confused by the differences in the descriptions by different observers, and still more so, perhaps, when he compares a larynx under observation with either of these descriptions.

This, then, is a part of French's description of the principles of laryngeal function in the production of tones. As he pointed out, no single description of the position of the vocal cords for the production of any given tone can apply for all larynxes or even for the same larynx under varying circumstances. Nevertheless, a general scheme of things persists, as originally described by French and, more lately, by others, including Guthrie,[5] Curry,[6] Pressman[7] and Russell.[8] A résumé of this "general scheme of things" is the purpose of this paper.

5. Guthrie, D.: Physiology of the Vocal Mechanism, Brit. M. J. **2**:1189 (Dec. 10) 1938.

6. Curry, R.: Mechanism of the Human Voice, London, Longmans, Green & Company, 1940.

7. Pressman, J. J.: The Action of the Larynx, J. Laryng. & Otol. **53**:672 (Oct.) 1938.

8. Russell, G. O.: (*a*) Physiological Cause of Voice Quality Difference, in Year Book, Carnegie Institution of Washington, 1938, vol. 37, p. 384; (*b*) 1933, vol. 32, p. 357; (*c*) 1929, vol. 29, p. 414.

THE VOCAL FOLDS OR TRUE VOCAL CORDS

The vocal cords are two in number, lying in a horizontal position, one on each side of the interior of the larynx, and situated about midway between the upper and lower laryngeal borders. Each is attached posteriorly to the vocal process of the arytenoid cartilage of the same side and passes anteriorly, converging toward its fellow, to be attached in front to the inner aspect of the angle of the thyroid cartilage, immediately alongside its fellow, almost at a common site. In all positions, therefore, except extreme adduction, the two cords form an acute angle of variable width, depending on the degree of abduction, the apex of which angle is located anteriorly.

Fig. 2.—Lateral view of a partially dissected larynx. The free margin of the cricothyroid membrane forms the "vocal ligament" of the true vocal cords (after Gray).

These structures are not fibrous bands, as one is apt to consider them and as their superficial appearance would lead one to believe. They are, on the contrary, composed chiefly of striped voluntary muscle covered by mucous membrane; each, however, includes within its substance a fibrous band of yellow elastic tissue known as the "vocal ligament." This ligament is essentially the upward continuation of the cricothyroid membrane[9] (fig. 2). The muscles which form the chief

9. Gray, H.: Anatomy of the Human Body, edited by W. H. Lewis, ed. 22, Philadelphia, Lea & Febiger, 1930, p. 1087.

bulk of the vocal cords are the inferior divisions of the thyroarytaenoidei (fig. 3). The true vocal cord is, on coronal section, roughly triangular in shape, with its flat surface above (fig. 4), and its inferomedial surface more or less concave. This gives the cord, on mirror examination, the appearance of a flat band, which, of course, it is not. The shape of the vocal cord, however, varies with its functional activity at the moment. The relatively sharp margin which constitutes the junction of the superior and more or less concave medial face represents the "free margin of the vocal cord."

The muscular structure of each true vocal cord, as I have indicated, is represented by the inferior division of a thyroarytaenoideus muscle. Each inferior division passes backward, to be inserted into the vocal process of the arytenoid cartilage and the adjacent anterolateral surface of

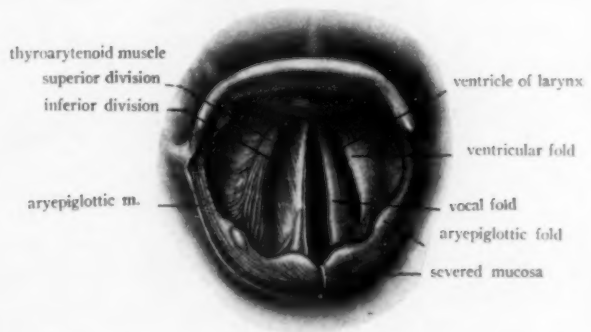

Fig. 3.—Superior view of the larynx. The mucosa has been removed on the left, exposing the muscular structure of the true and false vocal cords and the aryepiglottic fold. The inferior divisions of the thyroarytaenoidei constitute the chief bulk of the true vocal cords.

its main body. As this portion of the muscle continues backward from its origin, it contributes numerous and physiologically very important fibers which terminate near or at the free border of the vocal cord throughout its length. This portion of the muscle is sometimes given independent identity as the "vocalis muscle."

HISTOLOGIC STRUCTURES

Something of the histologic structure of the vocal cords has already been mentioned. The mucous membrane overlying the true vocal cords is peculiar in that its cellular structure is variable over different areas. Along the superior surface the mucosa is characterized by columnar ciliated epithelium, while over the region of its free border it changes

to a tougher stratified squamous epithelium (fig. 5), one type blending gradually into the other. The reason for this is obvious. The squamous-covered portion is subjected to severe trauma by the forceful and repeated contact of the opposite cord during phonation, whereas the remainder of the cord is not subjected to trauma. Near the inpocketing of the ventricle it requires cilia for the purpose of ridding itself of foreign matter and mucus globules which normally flow over this part of the cord. This portion is therefore composed of a stratified columnar ciliated epithelium. The mucous glands themselves are very few in

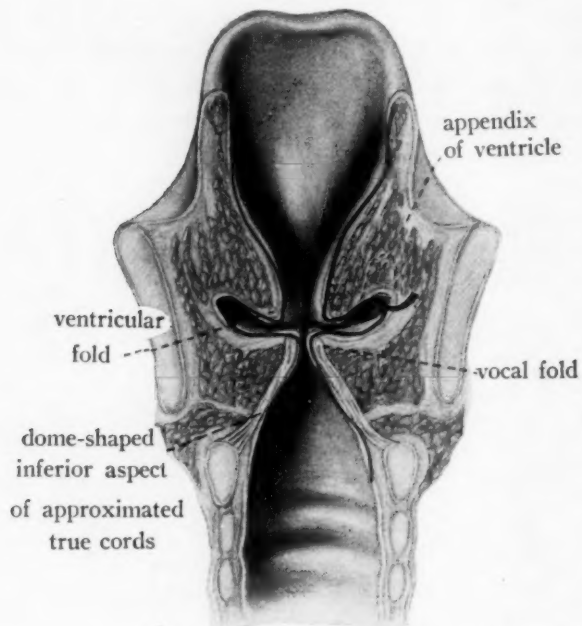

Fig. 4.—Coronal section of the larynx. The vocal fold, or true vocal cord, presents a flat surface above, a relatively sharp medial free margin and a concave inferior surface. The inferior surface of the approximated true vocal cords is therefore dome shaped.

the region of the free margin of the cords but become increasingly numerous along its superior surface as the cord blends into the ventricle, where they are numerous indeed. The glands are especially plentiful in the region of the saccule of the ventricle.

LUBRICATION OF THE VOCAL CORDS

At all times, but especially during phonation, the vocal cords are lubricated by a flow of thin mucus, which originates in the mucous glands of the entire larynx but chiefly in the ventricle and more espe-

cially in that upward, anteriorly placed projection of the ventricle known as the "saccule," the walls and surrounding areas of which are especially rich in mucous glands. A description of this function in an article by Freedman [10] is particularly clear and complete. The sacculus

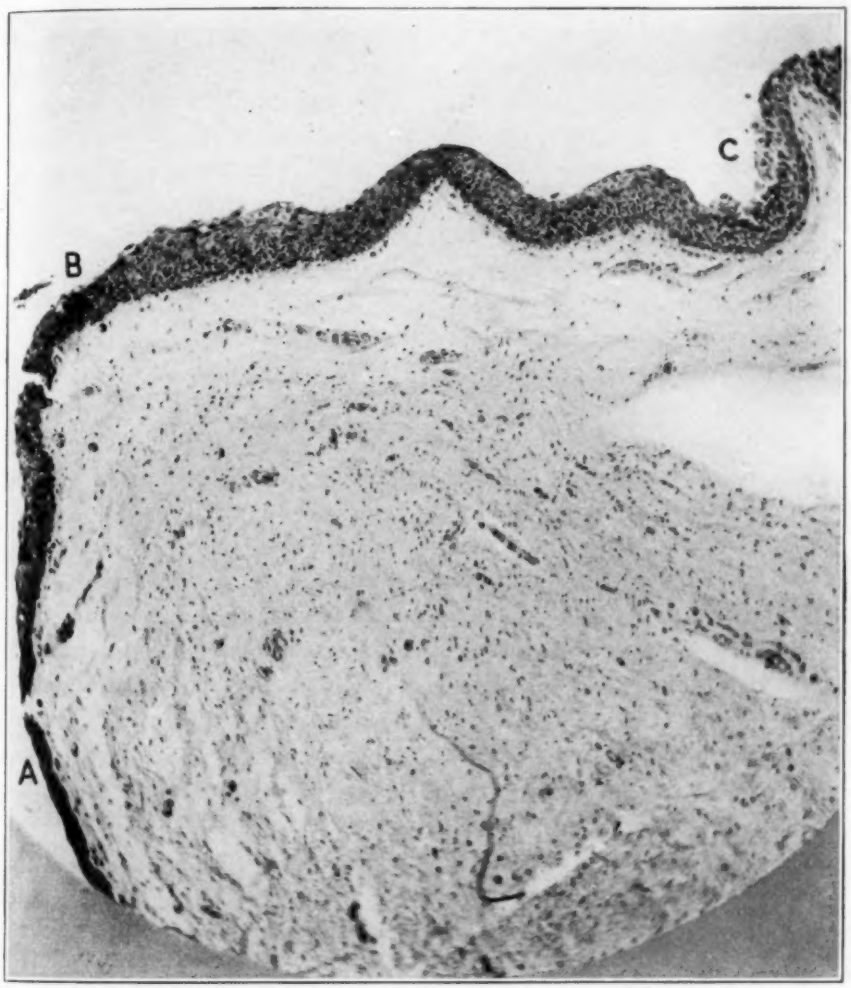

Fig. 5.—Epithelium of the true vocal cord (coronal section). The epithelium on the left at *A*, representing the medial surface, is narrow, squamous cell and almost cornified. As it reaches the free border at *B*, it is still squamous, but stratified. It becomes wider as it passes over the superior surface, blending into a stratified columnar ciliated type as, at *C*, it approaches the ventricle of the larynx.

10. Freedman, A. O.: Diseases of the Ventricle of Morgagni, Arch. Otolaryng. **28**:328 (Sept.) 1938.

laryngis is a reservoir of appreciable size, reaching upward for an average distance of half an inch (1.25 cm.) and often to the superior border of the thyroid cartilage or even much higher. It is therefore capable of holding in storage a large quantity of mucus, retained within its lumen by the action of two valvelike folds. Periodically, especially during phonation, the sacculus is emptied by the squeezing action of the surrounding compressor sacculi laryngis muscle of Hilton, which is really a muscular sheet composed of the combined fibers of the superior division of the thyroarytaenoideus and those few encircling fibers of the interarytaenoideus which pass anteriorly. While there is an almost constant flow of mucus over the vocal cords from the walls of the ventricles, a decidedly periodic mechanism of lubrication is nevertheless present as a result of this action of the sacculi, so that, as Freedman [10] has said, the mucus is poured over the vocal cords "in squirts." Thus during phonation there periodically appears over each vocal cord a collection of thin mucus, and its first appearance, regardless of the position of the patient, even when he is lying on his back so that the force of gravity would tend to produce the opposite effect, is always near the anterior commissure, at the point of exit of the saccule into the ventricle. Furthermore, the appearance of this jet of mucus, so far as I can tell, almost always occurs during the forceful contraction of the laryngeal structures, as in phonation, with the cords adducted, rather than during relative relaxation, as in abduction of the cords. This is very likely due to the increased compressor action of the "compressor sacculi" muscles during the more forceful stage of laryngeal activity. Undoubtedly, however, from time to time exceptions to this take place. When the collection of mucus appears at the anterior commissure, it spreads itself over the anterior segment of the approximated cords and then flows over them in waves from front to back, to reach the posterior limits of the cords, collecting, when excessive in amount, into a small pool at the posterior commissure. It then either is aspirated into the trachea when the cords open or overflows into the piriform sinuses. With the patient upright, it may well be that this flow of mucus in a posterior direction would not be so clearly demonstrable.

GENERAL PRINCIPLES OF PHONATION

Before considering in detail the physiologic aspects of phonation, several facts must be made clear. In the first place, the larynx can by no means be considered the organ of speech. Speech, as such, is entirely possible without a larynx, as evidenced by the fact that a person

who has had a total laryngectomy may well develop an acquired voice.[11] Under ordinary circumstances the larynx can, however, be considered as the "organ of sound production," but in the stricter sense even this is not entirely true. The sounds which emanate from the larynx are not at all the sounds one hears emanating from the mouth.[12] They are, on the contrary, feeble and without resonance or "color," requiring modification in the upper and lower respiratory tract before becoming what are known as vocal tones.[8] Laryngeal tones, however, present the element of fundamental pitch in its final form, but this is practically the only element of the voice which is solely dependent on laryngeal function. Tones produced by the human larynx are not pure tones, or "fundamentals," but consist of these plus overtones, or "partials."[13] The fundamental pitch, as well as overtones, originates in the vocal folds of the larynx, but in all likelihood the latter, too, are modified by structures of the upper respiratory tract. Variations in the pitch of these overtones identify a given tone as coming from one larynx, as compared with that coming from another,[12] and contribute in part to the production of a pleasant or an unpleasant voice. For instance, if very high overtones are accentuated, the tone produced is shrill and harsh.

The larynx, therefore, is a tone-producing organ furnishing raw material in the form of sound, which is modified into the final form of the human voice by various resonating chambers above and below the larynx itself. This modified sound is ultimately converted into speech by the action of the pharynx, tongue, lips, palate and related structures. However, I point out again, for the sake of emphasis, that this conversion of sound into speech is in no way dependent on the larynx, for sound reaching these upper respiratory structures from any other source, such as the esophagus in laryngectomized persons, can similarly be converted into speech. Under the latter circumstances, however, adequate variation in pitch is lacking, a phenomenon which is recognizable as a laryngeal function.

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11. (*a*) Morrison, W. W., and Fineman, S.: Production of Pseudovoice After Total Laryngectomy, Tr. Am. Acad. Ophth. **41**:631, 1936. (*b*) Jackson, C. L.: The Voice After Direct Laryngoscopic Operations, Laryngofissure and Laryngectomy, Arch. Otolaryng. **31**:23 (Jan.) 1940. (*c*) Levine, N.: Speech Following Total Laryngectomy, privately published reprint, Broadwood Press, 1940. (*d*) Schall, L.: Psychology of Laryngectomized Patients, Arch. Otolaryng. **28**:581 (Oct.) 1938.

12. Negus, V. E.: Evolution of the Speech Organs of Man, Arch. Otolaryng. **28**:313 (Sept.) 1938.

13. Hala, B.: L'analisi oscillografica della voce laringea nel riguardo alla formazione delle vocali, Valsalva **14**:332 (July) 1938.

THEORIES CONCERNING THE PHYSICAL PRINCIPLES INVOLVED IN THE PRODUCTION OF TONES AT THE LARYNX [14]

Tones produced at the human larynx result from the expiration of air through a narrowing of the airway. This constriction consists in the approximation of the vocal bands, and the walls of the airway are therefore elastic. The cords themselves are capable of varying in length, tension and contour, thereby regulating the size of the laryngeal lumen as well as undergoing vibratory movements. As Guthrie [5] pointed out, the laryngeal muscles do not produce the vocal note. Phonation is an aerodynamic phenomenon, in which the muscles merely adjust and hold the cords in a certain position, at a certain tension and with a certain shape. The expiratory air current from the lungs does the rest.

The mechanism by means of which vibratory movements of the vocal cords result in the production of sound is the subject of much discussion. Several theories are in vogue. Of these, the theory of the likeness of the larynx to a musical reed instrument is widely held. Regardless of the accuracy of any theory concerning the mechanics of vibrating vocal cords, the fundamental fact is that air under pressure passing through the narrow glottic chink produces a tone by stimulating the vocal bands to vibration. Changes in the contour of this chink, plus alterations in the elasticity of the margins, as the Jacksons [15] pointed out, produce variations in pitch.

The Reed Theory.—According to the reed theory,[16] tone production and pitch variation at the larynx are explained by a mechanism in which the vocal cords function in much the same way as the reed of any wood-wind musical instrument containing a reed mouth piece. The reeds (or vocal cords in this case) come together momentarily and more or less close off the outlet valve. They then open, allowing the air to escape in puffs. This process, repeated rapidly, represents the vibrations of the already approximated vocal cords and is not to be confused with simple abduction and adduction. In this manner the stream of air is cut off periodically by approximation of the cords. It is this rapid

14. For a comprehensive description of the physical principles involved in the mechanism of tone production within the larynx and a comparison with that of various musical instruments, see an article by Jackson (Jackson, C.: Myasthenia Laryngis, Arch. Otolaryng. **32**:434 [Sept.] 1940), published too recently to be considered fully in this presentation.

15. Jackson, C., and Jackson, C. L.: The Larynx and Its Diseases, Philadelphia, W. B. Saunders Company, 1937, p. 21.

16. Schatz, H. A.: The Art of Good Tone Production, Laryngoscope **48**:656 (Sept.) 1938. Wessely, E.: Die Veränderungen der Stimme bei Larynxerkrankungen, Wien. med. Wchnschr. **88**:574 (May 21) 1938. Guthrie.[5] Negus.[12] Russell (footnote 8 *b* and *c*).

escape of puffs of air which produces the sound. There can be no doubt that the expired air escapes in puffs; nor can there be any doubt that the rapid escape of air in puffs can result in the production of sound, since this phenomenon can be observed readily in the earlier models of the siren constructed with a circular metal plate perforated around the margin and rotated in front of a forceful stream of air. Variations in pitch, according to the "reed theory," result from the action of the vocal cords in much the same way that pitch is changed by the action of the mouth of a bugler, i. e., by enlarging or diminishing of the size of the orifice, variation in its rigidity and changes in its shape and by changes in the force of expired air. In the action of the vocal cords, proponents of this theory see a striking resemblance to the action of a bugler's or trumpeter's lips.[5]

However, it is impossible to conceive of the larynx entirely as a reed instrument, since in the latter, changes in pitch occur as the result of the action of resonating chambers, whereas there can be no doubt that in a human being variations in pitch result, at least in part, from changes in the length, tension and contour of the vocal cords themselves.

Because of objections to the "reed theory," an explanation likening the action of the vocal bands to the action of the strings of such musical instruments as the violin has its supporters.[17] It is known as the "vibrating string theory."

The Vibrating String Theory.—This concept provides that the vocal cords are enabled to produce tones and to vary their pitch in a manner analogous to that of a violin string. In a previous paper I [18] have discussed certain prevailing arguments in support of this view. The source of stimulation in the larynx is the power of the expiratory air blast, and in the violin, the movement of the bow across the string; but, regardless of the type of stimulation, the end result, namely, the production of vibrations, is the same. Under these circumstances pitch can be varied in the larynx by increasing the tension of the cords, just as the pitch of a violin string is elevated by tightening the string when one tunes the violin. That a tone so produced by muscular vibrations becomes elevated when tension of the muscles is increased has been proved by Negus.[19] Because of the limits of elasticity inherent in any vibrating string, elevation in pitch can be carried only so far by this act of increasing the tension by a process of elongation, or tightening. When this limit has been reached, further elevation in pitch can, however, be

17. Trendelenburg, W., and Wullstein, H.: Investigations of Vocal Cord Vibration, abstracted, J. Acoust. Soc. America **12**:306 (Oct.) 1940.

18. Pressman, J. J.: Physiology of the Larynx, Laryngoscope **49**:239 (April) 1939.

19. Negus, V. E.: Mechanism of the Larynx, St. Louis, C. V. Mosby Company, 1929.

accomplished as the result of a phenomenon akin to fingering a violin string on the finger board and thus shorting the length of string which is free to vibrate. In the larynx this functional foreshortening takes place by the approximation of portions of the vocal cords against each other, beginning posteriorly. The segments in contact with each other are not able to vibrate, so that each contacting segment is said to be "damped" by the other. As a result of this, the remaining segment, which is free to vibrate, becomes functionally shorter and a corresponding elevation in tone is produced. Proponents of this theory point out, in addition, that as greater lengths of the cords come into contact with each other, the hiatus between the cords becomes correspondingly smaller and smaller. The diminution in size of the outlet further tends to elevate the pitch, according to those principles of the physics of sound relating to the passage of air under pressure from a large cavity through a narrower orifice. This effect is seen to good advantage in peanut whistles, for example, in which a tone of very high pitch is produced, because of the minute orifice.

Few authorities can agree on the exact physical principle involved in the production of sound at the larynx. Perhaps no one single principle is at work, but rather, several. The larynx of mammals is unique and likely has no exact counterpart in man-made instruments. At any rate, a more detailed discussion of the complex problems in physics relating to this issue has no place here, and even if such a discussion were to be entered on, it would of necessity have to be undertaken by others who are better qualified to do so.

OBSERVATIONS ON THE PHYSIOLOGIC ACTION OF THE LARYNX DURING THE PRODUCTION OF SOUND

General Considerations.—Regardless of whether or not one is able to understand or to explain to the satisfaction of others the exact physical method involved in the production of tones at the larynx, a certain amount of accurate information is available concerning the physiologic changes taking place as tones of varying pitch and intensity are produced. Since the time of French,[2] laryngologists have been familiar, from direct observation, with those variations in the vocal cords and related structures which accompany the phenomena of tone production in a variety of its phases. It is, too, quite possible to theorize more or less accurately on the mode of muscular action which brings them about.

It must be repeated, as French [2] pointed out, that it is not at all possible to describe any particular appearance or configuration of the larynx, more particularly of the vocal cords, as resulting in the produc-

tion of any given tone. Variations among persons in the length, breadth, general outline, degree of elasticity and tension of the vocal cords, as well as in habits concerning the degree of expiratory force utilized in producing tones, make it impossible for any given tone to be produced in different subjects by exactly the same mode of laryngeal action. Even two persons with exactly the same range and with voices strikingly similar are not likely to produce a particular tone in exactly the same way. If, for example, one uses slightly less expiratory force than the other in producing an exactly equivalent tone, then this diminished air pressure must be compensated for by an increased approximation, or tension, of the vocal cords. It may even be necessary, for instance, to bring into contact the posterior tips of the cords for the purposes of dampening, whereas this did not prove to be necessary for the person utilizing a greater force of expired air. Under these circumstances equivalent tones are accompanied by an entirely different appearance of one larynx as compared with that of another. Yet, not only may these differences in expired air pressure and laryngeal configuration result in the production of the same tone, but also the volume of the sound thus produced may be exactly the same, on account of differences in the resonating chambers of the two persons and other indeterminate characteristics, even though expiratory pressure and the contour of the vocal cords remain the same.

One can, therefore, never ascribe a particular laryngeal picture to the production of any given tone. However, certain general principles apply, and with respect to any larynx it is possible to prognosticate within certain limits what changes will take place in that larynx as one begins at the lowest tone which the particular larynx can produce and ascends the scale to reach its highest possible tone.

A thorough understanding of these mechanics requires a rather intimate knowledge of the anatomy of the intrinsic muscles of the larynx. I hope that reference to the accompanying illustrations will serve as sufficient review, since it is not possible to enter into a complete detailed anatomic description. It is necessary to limit this portion of the discussion to a résumé of the action of these muscles. Unfortunately, it has been the custom to think of laryngeal muscular activity in terms of individual pairs of muscles or of groups, such as "abductors," "adductors" and "internal tensors." This is a sound point of view as far as it goes but not altogether satisfactory. One must think rather of what goes on in the vocal cords and their associated structures, picturing in one's mind the muscular interplay of the various groups of muscles responsible for each movement, and place less stress on the individual action of each muscle. Only in this way is it possible to conceive of the true picture of the larynx as a physiologically active organ rather than

as a dissected anatomic specimen. It is this grouping of the actions of these muscles to account for any given movement, rather than any new thoughts on the action of any individual muscle, that seems to warrant devoting some time and space to the sketchy descriptions which follow.[20]

The fundamental mechanics of laryngeal physiology in so far as phonation is concerned is that the vocal cords abduct away from the midline when vocal sounds are not being produced and adduct toward it, after which movement they undergo variations in length, in tension and in approximation one to the other, in order to produce the various tones. Such movements, as a part of phonation, can be divided into two main groups, i. e., those dependent on rotation and approximation of the arytenoids and those occurring independently of or in addition to arytenoid movements.

The first step in the process of producing a tone at the larynx is the approximation of the vocal cords to the midline (adduction), which is essentially due to arytenoid rotation, followed by an increase in tension of the cords and their separation against this tension to permit the escape of air and the vibratory movements. The last-named phenomena occur for the most part independently of the arytenoid movements. Each of these processes will be described in turn.

MOVEMENTS OF THE VOCAL CORDS DEPENDENT ON ROTATION OF THE ARYTENOID CARTILAGES

The two vocal cords are attached anteriorly at relatively the same fixed point on the thyroid cartilage. Each is attached posteriorly to a movable point, the vocal process of the corresponding arytenoid cartilage. From this anatomic setup it can be readily understood that certain movements of the vocal cords are dependent on, and take place as a result of, movements of the arytenoid cartilages. Although abduction is not an essential part of phonation, it will, for the sake of completeness, be described first.

Abduction.—The principal movements (but not the only movements) of the arytenoid cartilages are a clockwise and counterclockwise rota-

20. I must point out, however, that many of these anatomicophysiologic observations are largely conjecture, based partially on my own observations of the anatomy of the organ and partially on the writings of such time-honored authorities as the authors of the usual standard texts on anatomy (Gray, Piersol and Cunningham) and, finally, on observations of pathologic laryngeal activities resulting from paralysis of the various laryngeal nerves. They are in no sense, however, intended to be authoritative final statements based on experimentation, but rather represent a picture which to me has proved satisfactory in explaining certain physiologic phenomena which are known to take place. I cannot hope that such descriptions of muscle action as I have included will carry weight if and when future experimental work on the function of the muscles presents evidence to the contrary. To date, however, no such evidence has been produced.

tion, which takes place on the long axis of the cartilages, using as a fixed base their articulation on the facet of the cricoid cartilage. If this movement begins with the vocal process at the midline and rotation takes place in an outward direction, the corresponding vocal cord is carried outward, but at an angle, because of its fixation anteriorly. Since such rotation in an outward direction takes place in both arytenoids simultaneously (the outward rotation of the left cord being counterclockwise and that of the right clockwise), the result is that the vocal cords are separated from each other, forming an angle, the apex of which is situated anteriorly at the fixed point of insertion of the cords on the thyroid cartilage. The space within this angle is the rima glottidis, or glottic chink. Its posterior boundary is the mass of the interarytenoid muscles covered by mucosa. The width of the angle varies with the degree of abduction which has taken place. This outward rotation of the arytenoid cartilages with the resulting abduction of the vocal cords occurs chiefly from the action of a single pair of muscles, the posterior cricoarytenoids, and partially from that of the interarytenoids. The origin of the former muscles is at a fixed point on the posterior face of the cricoid cartilage, the fibers passing laterally to the muscular processes of the arytenoids. The contraction of these fibers results in the rotation of the arytenoids outward, toward the fixed point of origin of the muscles, with resultant abduction of the cords.

The bilateral cortical innervation of these muscles is such that each posterior cricothyroid muscle always contracts (and relaxes) in exactly the same way and at the same time as its fellow, so that the resultant rotation of each arytenoid is simultaneous with and roughly equal to the corresponding movement of the other. It is under no circumstances (except when one vocal cord is paralyzed) possible for one muscle to function without a relatively equal and similar reaction taking place on the opposite side. The movements of the two sides of the larynx are, therefore, always alike, allowing, of course, for minor variations, and this is true of the action of paired muscles throughout the larynx.

Adduction.—The cords having been abducted and the chink of the glottis opened, one must next consider the method of closure, by means of which the cords, throughout their length, are completely approximated to one another at the midline and begin to assume what is known as "the position of phonation." Several pairs of muscles are involved, each of which carries the closure to a position nearer completion or at least lends its strength to the adductor movement.

The first of these are the cricoarytaenoidei laterales. From their more anterior insertion on the rim of the cricoid cartilage the fibers run backward to the muscular process of the arytenoid. Shortening of the muscle fibers by contraction toward their more anterior fixed point of

origin reverses the action of the cricothyroidei postici and turns the movable arytenoids in an inward rotary direction, so that the vocal processes and, therefore, the vocal cords are approximated toward the midline. The antagonistic cricoarytaenoidei postici relax, according to Sherrington's laws of reciprocal innervation.[21] The closure resulting from the action of these muscles is, however, only partial, a rather broad longitudinal hiatus still persisting throughout their length (fig. 6). The tension of the cords has not been increased appreciably. Further closure takes place by contraction of the interarytaenoidei.

The interarytaenoidei decussate across the posterior commissure, from the posterior aspect of one arytenoid to that of the other. The contraction approximates one arytenoid to the other by a sliding move-

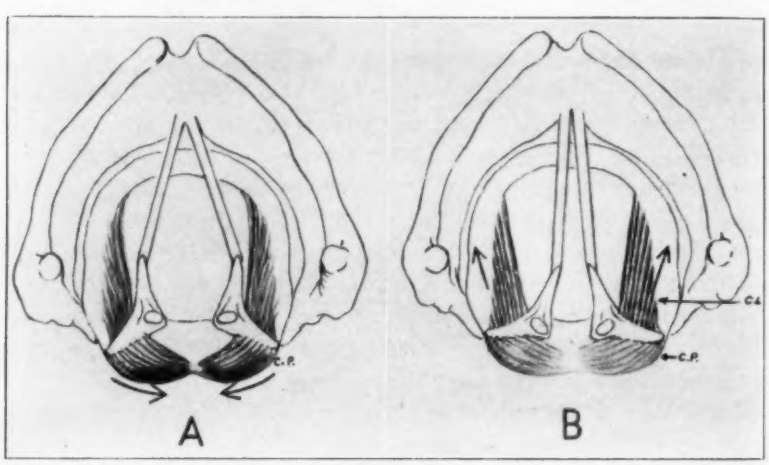

Fig. 6.—Antagonistic action of the cricoarytaenoidei postici and the cricoarytaenoidei laterales. At *A* the contraction of the cricoarytaenoidei postici (*C.P.*) has rotated the muscular processes of the arytenoids toward the midline, so that the vocal processes abduct away from it. The cords therefore open. At *B* the cricoarytaenoidei laterales (*C.L.*) contract, reversing the process and closing the cords. Note that complete closure has not taken place. This represents the first stage in the assumption of the position of phonation.

ment (fig. 7, *A* and *B*). This action furthers the movement toward complete approximation by closing the posterior commissure. Notice, however (fig. 7 *B*), that this sliding movement still leaves the vocal processes of the arytenoids in a "toed out" position, requiring further inward rotation for their complete closure. This final stage of inward rotation is accomplished by the action of a third pair of muscles, the complex thyroarytaenoidei, the fibers of which, including both the

21. Starling, E.: Human Physiology, ed. 3, Philadelphia, Lea & Febiger, 1920, p. 355.

superior and the inferior divisions, pass from the wedge of the thyroid cartilage anteriorly to the muscular and vocal processes and the body of the arytenoids posteriorly. The contraction of these groups of fibers, especially those of the muscular processes, rotates these processes toward the midline, thus enhancing a similar action of the other adductor muscles, and completes the approximation of the arytenoids. The approximation, in turn, almost but not entirely completes the closure of the vocal cords one to the other (fig. 7 *C*). A hiatus still remains anteriorly.

As a result of the action of this triad of muscle pairs on the arytenoids, the vocal cords have, then, become closely approximated to one another except for their anteriormost segments, and a certain degree of tension has developed. Their complete approximation requires the action of an additional pair of muscles, an action which, in addition,

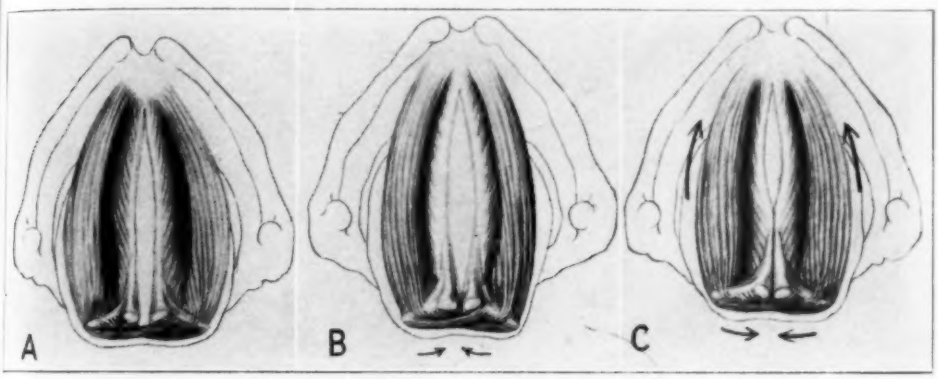

Fig. 7.—Closure of the posterior commissure by the action of the interarytaenoidei and the thyroarytaenoidei (second state of closure for the position of phonation). *A*, position prior to closure. *B*, approximation of the arytenoid cartilages to the midline as a result of action of the interarytaenoidei. Note that the vocal processes remain "toed out." *C*, the thyroarytaenoidei (aided by the cricoarytaenoidei, which are not demonstrated) further rotate the arytenoid cartilages to the midline, thus bringing the vocal processes together. The posterior commissure is now closed completely. Note that a gap still persists anteriorly.

increases the tension to a point necessary for normal phonation. These muscles act independently of any rotary movements of the arytenoids and are therefore considered independently.

MOVEMENTS OF THE VOCAL CORDS NOT DEPENDENT ON ROTATION OF THE ARYTENOIDS

Further and complete closure of the vocal cords and the assumption and maintenance of the required degree of tension depend on the action of the fanlike cricothyroidei muscles. From a point of origin anteriorly

and below, on the lateral face of the cricoid cartilage, the fibers spread out posteriorly and upward to the inferior cornu and the lower margin of the ala of the thyroid cartilage. Contraction of this pair of muscles completes the closure by elongating the elastic vocal cords, by a process

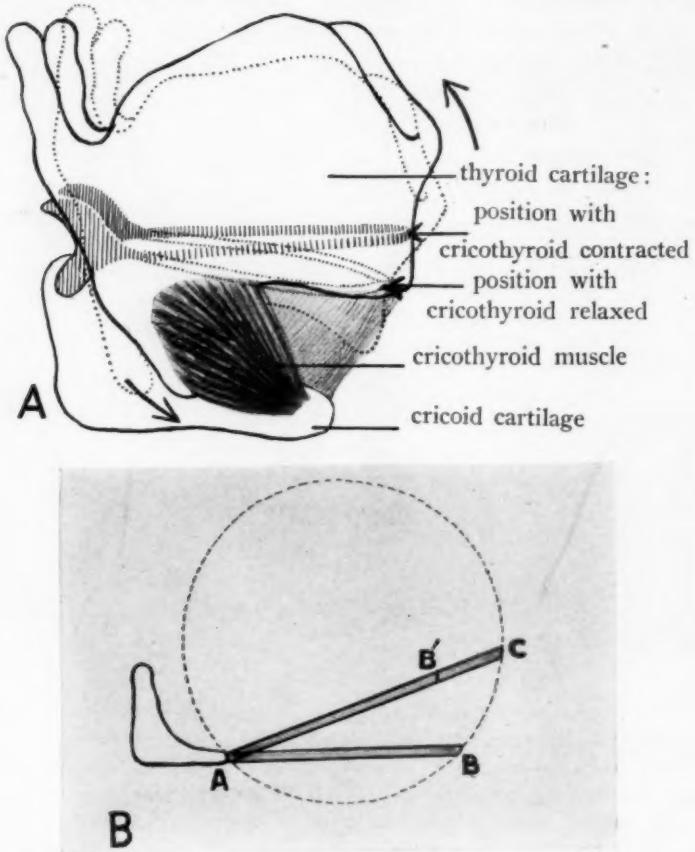

Fig. 8.—*A*, action of the cricothyroidei in bringing about elongation and the final stage of closure of the vocal cords. The contracted cricothyroid muscles tilt the thyroid cartilage, widening the cricothyroid gap and rotating the anterior insertion of the vocal cords upward over the arc of a circle. The resulting increase in length closes the cords completely. *B*, diagram illustrating the mechanism of elongation of the vocal cords resulting from upward tilting of the thyroid cartilage. *A* indicates the posterior attachment of the vocal cord to the vocal process of the arytenoid cartilage; *B*, the anterior attachment of the vocal cord to the thyroid cartilage. *AB*, the length of the vocal cord prior to contraction of the cricothyroidei muscles. *BC*, the arc of rotation of the anterior wedge of the thyroid cartilage resulting from contraction of the cricothyroidei. Therefore, *AC*, the length of the vocal cord after the contraction of the cricothyroidei. *AB* is equal to *AB'*. Therefore, *B'C* indicates the increased length of the vocal cord resulting from the upward tilt of the thyroid cartilage due to contraction of the cricothyroidei muscles.

of "taking up slack." This elongation is accomplished as follows: The thyroid cartilage is capable of superoinferior rotation on its cricothyroid joints. When the posterior fibers of the cricothyroid muscles exert their downward pull on the ala and inferior cornua of the thyroid, this cartilage undergoes a rotary movement which tends to pull the posterior portion of the cartilage downward and forward, as a result of which the anterior portion, to which the anterior extremities of the vocal cords are attached, rotates upward. This rotary upward movement of the anterior insertion of the vocal cords on the thyroid cartilage increases the distance between this point and the site of the posterior insertion of the cords on the arytenoids (fig. 8). As a result of this increase in intralaryngeal diameter, the cords become elongated and are brought into complete approximation in the same fashion that the sides of a

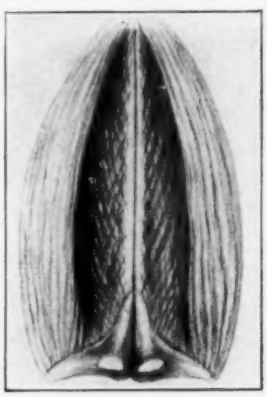

Fig. 9.—Final stage of complete closure, elongation and increase of vocal cord tension resulting from the action of the cricothyroidei. Figures 6 and 7 represent the previous stages.

flaccid rubber band are approximated when the band is stretched and placed under tension.

Not only are the cords elongated by this action of the cricothyroidei, but the action of this pair of muscles superimposed on that of the previously mentioned lateral cricoarytaenoidei, interarytaenoidei and certain fibers of the thyroarytaenoidei completes the closure of the vocal cords and brings them into a high state of tension (fig. 9).

There can be no doubt that such elongation of the vocal cords takes place, although this has not always been conceded. Motion picture photographs of the larynx (fig. 10) taken by members of the staff of the Bell Telephone Company Laboratories [22] show this clearly. They demonstrate the degree of elongation and stretching of the vocal cords

22. High Speed Motion Pictures of the Human Vocal Cords: Circular, New York, Bell Telephone Company Laboratories, 1939.

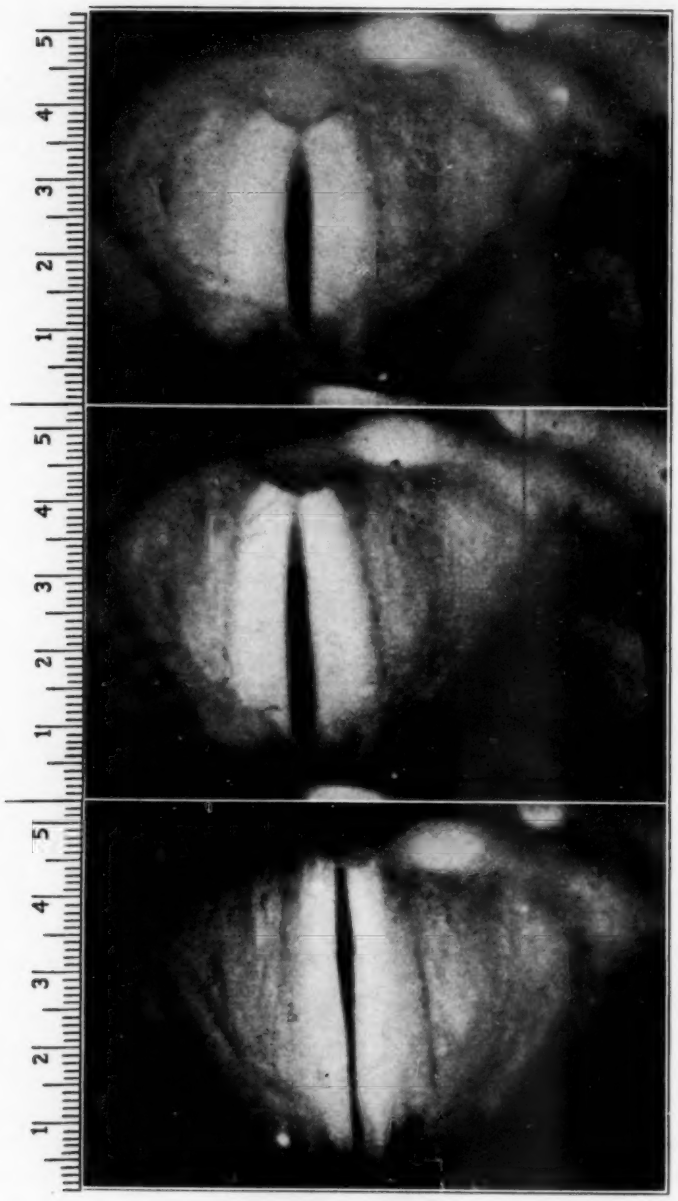

Fig. 10.—Elongation of the true vocal cords (Bell Telephone Company Laboratories [22]). The photographs represent three stages in the gradual transition (top to bottom) from a lower tone (about 120 double vibrations) to a higher one (about 300 double vibrations). The magnification is the same in all instances. There can be no question that elongation of the vocal cords has taken place.

which can take place. In figure 10 the cords are not approximated completely, because they have already been pulled apart against tension to allow vibrations to take place. The illustration is intended to demonstrate solely the increase of the intralaryngeal diameter which can take place.

It must be pointed out, however, that for this elongation and stretching to take place, the cricoid cartilage must be fixed in a posterior position and the arytenoid cartilages fixed on it, in order to prevent their forward displacement from the pull of the anteriorly placed adductor muscles. If such forward displacement of the cricoid cartilage itself or of the arytenoids articulating on it did take place, the vocal cords, as a result, would become foreshortened and flaccid instead of lengthened and tensed, which would defeat the entire purpose of the phonatory mechanism. It is therefore necessary to describe this posterior fixation.

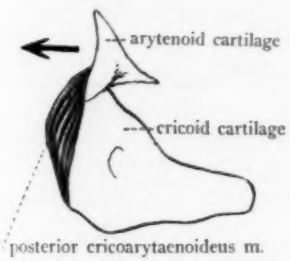

Figure 11

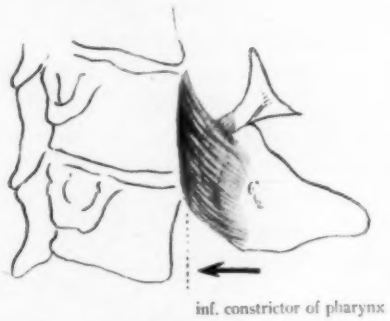

Figure 12

Fig. 11.—Posterior fixation of the arytenoid cartilages on the cricoid. The cricoarytaenoidei postici, in a state of rigid contraction, prevent a forward sliding of the arytenoid cartilages on their cricoidal facets and thus establish a firm posterior site of fixation for the vocal cords. This enables the cords to be elongated and placed under tension. Otherwise they would foreshorten and become flaccid.

Fig. 12.—Posterior fixation of the cricoid cartilage by the inferior constrictor of the pharynx. Posterior fixation of the cricoid cartilage is necessary to prevent a forward displacement of the cricoid and arytenoid cartilages as a unit. Such displacement would result also in flaccidity and foreshortening, just as though the arytenoid cartilage alone had been moved forward (fig. 11).

POSTERIOR FIXATION OF THE ARYTENOIDS AND THE CRICOID DURING ADDUCTION

It can be observed easily, as I have just indicated, that the anteriorly placed adductor muscles, in addition to the other actions already described, pull on the arytenoids from a point of origin anterior to them. Such action tends to pull the arytenoids forward, and unless a

compensatory mechanism were brought into play, to fix the arytenoids in a posterior position, the forward displacement would shorten and relax the vocal cords. The necessary posterior fixation and counterbalancing backward pull on the arytenoids, fixing them to the cricoid and preventing their displacement anteriorly, can result from the action of the cricoarytaenoidei postici. These, arising as each does from a broad base on the posterior aspect of the cricoid cartilage and inserting on the posterior aspect of the arytenoid, tend, while in a state of contraction, to prevent the arytenoids from sliding forward on their cricoidal facets (fig. 11). This, however, would not prevent a forward displacement of the arytenoids by the adductor muscles unless the cricoid itself were fixed posteriorly, for if it were not, then the forward movement would take place with the cricoid and arytenoids moving as a unit. The required posterior fixation of the cricoid is effectively carried out by the action of the inferior constrictor of the pharynx, which arises from the sides of the cricoid and thyroid cartilages. The point of origin of this muscle on the cricoid is between the cricothyroideus muscle in front and the articular facet for the inferior cornu of the thyroid cartilage behind. The fibers spread medially and backward to be inserted, by a raphe, on the posterior wall of the pharynx. This raphe is, in turn, closely attached to the vertebral column. Thus, a very strong effective backward pull is exerted on the cricoid, which prevents its being pulled forward and allows the adductor group to exert its pull in the anterior direction without displacing the cricoid (fig. 12).

ANTERIOR FIXATION OF THE THYROID CARTILAGE DURING ADDUCTION

If the vocal cords are to be kept tense and on stretch during adduction, not only must the cricoid and the arytenoid cartilages be held firmly, so as not to slide forward, but the anterior site of insertion of the cords in the angle of the thyroid cartilage must be firmly fixed anteriorly, so as not to be displaced backward. This anterior fixation of the thyroid cartilage is accomplished for the most part by the cricothyroid muscles. If the cricoid cartilage is firmly fixed by the inferior constrictor just described, then contraction of the more nearly horizontal fibers of the cricothyroid muscles tends to pull the thyroid cartilage forward, or at least to stabilize it in an anterior position (fig. 13).

By this fixation of the thyroid cartilage anteriorly and of the cricoid and arytenoid cartilages posteriorly the cords are kept elongated and under sufficient tension so that the incidental and unavoidable action of the anterior pull of the adductors and other muscles on the arytenoid cartilages as they open and close the cords will not, as a secondary phenomenon, result in foreshortening and relaxation of the vocal cords.

INDEPENDENT MOVEMENTS INTRINSIC WITHIN THE VOCAL CORDS

I have already outlined the method by means of which the cords are completely approximated or adducted and maintained under tension, as a necessary preliminary to the establishment of the vibrations of normal phonation. From a study of slow motion pictures (fig. 14) it can be established readily that such a preliminary, almost complete adduction takes place just before the initial movement of the vibratory cycle. From this adducted position finely detailed intrinsic movements of the cords occur independently of any action of the arytenoids, which

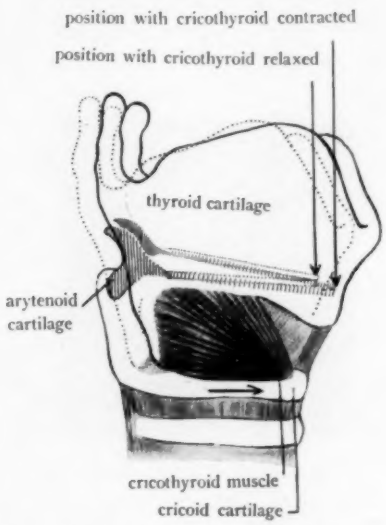

Fig. 13.—Anterior fixation of the thyroid cartilage by the cricothyroid muscles. For elongation and tension to be established, the site of the anterior insertion of the vocal cords must be maintained in an extreme forward position, just as the site of the posterior insertion of the cords into the arytenoids must be held backward. If the thyroid cartilage were to be displaced backward, foreshortening and flaccidity would result. This is prevented by the forward pull of the cricothyroid muscles.

remain motionless and fixed in closed apposition as these intrinsic movements of the vocal cords occur. The latter are really movements of segments of the cords instead of movements of the cords as a whole. They are due almost entirely to the action of certain fibers of the internal portions of the thyroarytaenoidei muscles and consist of a pulling apart, against tension, of the length or an equal portion of the length of each vocal cord, to establish the final pattern in which the vibrations will take place. Groups of these fibers have been described as individual

muscles, such as the "aryvocalis," but actually they can all be considered as parts of the thyroarytaenoideus.

The histologic anatomy of the course of the various fibers or groups of fibers of that portion of the thyroarytaenoidei which makes intrinsic movements of isolated portions of the vocal cords possible is not well known. That certain of these fibers attach themselves to the fibrous portion of the cord seems to be certain.[23] Their arrangement, however, is doubtful, and one can surmise a plan of distribution of fibers, not on the basis of any anatomic evidence, but rather by what takes place physiologically. Certain movements of the vocal cords seem to suggest that a definite plan of distribution is present. If this schematic arrangement which I describe does not hold true in the strict anatomic sense, it at least serves to provide a theoretic system of muscular mechanics well able to account for those variable intrinsic movements

Fig. 14.—Preliminary closure of the vocal cords under tension, immediately prior to their being pulled apart and undergoing vibration. A photographic demonstration of the stage of closure shown in figure 11, from a slow motion picture sequence.

of the cords which are known to take place [5] in the production of the various tones. It seems that the following three groups of fibers may be present (fig. 15):

1. Fibers arising anteriorly in the thyroid cartilage and passing posteriorly more or less parallel to the general direction of the cords, to terminate at different levels (fig. 15 *A*). The innermost fibers are very short and terminate in the anterior portion of the cords. The more lateral fibers are longer, inserting farther and farther posteriorly until the most lateral ones extend from the anterior point of origin on the thyroid cartilage to the posterior extremity of the cords near the arytenoids or into the vocal processes themselves.[23]

23. Piersol, G. A.: Human Anatomy, edited by G. C. Huber, ed. 9, Philadelphia, J. B. Lippincott Company, 1930, pp. 1825-1826.

2. Fibers arising at various points within the substance of the vocal cord and conus elasticus (of which the former is a part) and passing backward to insert on the body and vocal process of the arytenoid cartilages (fig. 15 *B*).

3. Fibers arising here and there in the substance of the cords and extending backward for varied distances to insert on the same cord at a more posterior point (fig. 15 *C*).

None of these three groups can be recognized as an entity, since a great deal of intertwining of the fibers of each group with those of the others takes place.

The action of these groups of fibers is on the cord and is probably as varied as the arrangement of the fibers is complex. Their most

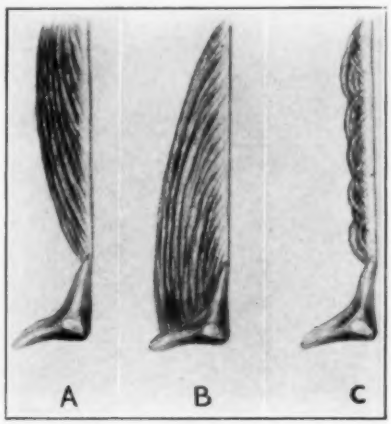

Fig. 15.—Theoretic arrangement of those groups of fibers of the inferior division of each thyroarytaenoideus muscle which form a portion of the true vocal cord (aryvocalis muscle).

important action in phonation seems to take place after the cords have been strongly and completely approximated and tensed by the adductor group. When this tension and adduction have been accomplished, it then becomes necessary to reopen the cords by some means which permits them to retain their relatively rigid elasticity. The action of these internal fibers of the thyroarytaenoideus muscle is admirably suited for the purpose. Starting with the arytenoids tightly closed and the vocal bands on stretch and closely adherent, those internal fibers of the thyroarytaenoidei which arise on the thyroid cartilage and insert into the cordal structure contract. They thus pull apart, against the tension of the adductors, those portions of the vocal cords into which they insert (fig. 16). This action takes place in much the manner that an archer draws back the tense bowstring of his bow, the arm of the archer rep-

resenting the muscle fibers of the thyroarytaenoideus muscle and the taut bowstring representing the tensed vocal cord. When the pull of these fibers is released, the cord returns sharply to the midline, corresponding to the release of the bowstring by the archer. The length of cord pulled apart is determined by the number of fibers of this portion of the thyroarytaenoideus muscle which are called into play. The manner in which such selective action of certain fibers takes place was described by Kistler.[24] The variation in the length of cord pulled apart is important from the standpoint of variations in pitch. This will be described later.

Those fibers of the thyroarytaenoideus muscle arising in the vocal process of the arytenoid and extending forward to be inserted into the

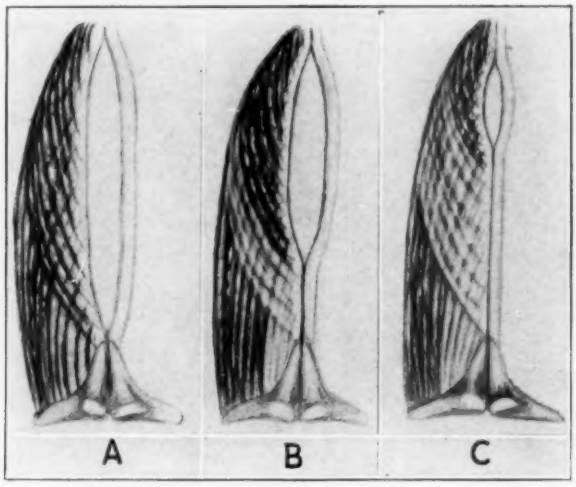

Fig. 16.—Action of the aryvocalis fibers of the inferior division of each thyroarytaenoideus muscle. The outward pull of various groups of fibers, one supplementing the other, determines the length of vocal cord which will be separated from its fellow and thus brought into a position of vibration. *A*, *B* and *C* represent three stages of separation, each producing a higher tone. The darker-shaded muscle fibers are in each case exerting the outward pull on the muscle band.

cord at different points have the same action, supplementing that just described, except that the pull comes from a posterior instead of an anterior point. The chief reason for believing that there are such fibers is that if a segment of the cord is to be bowed laterally, as actually occurs, instead of anterolaterally, a compensatory group of fibers must exert a posterior pull against the action of any group that might arise anteriorly.

24. Kistler, K.: Physiologische Betrachtung bei der Behandlung von Rekurrenslähmungen, Schweiz. med. Wchnschr. **68**:44 (Jan. 8) 1938.

The fibers arising in one portion of a cord and inserting in another further supplement this action and give the margins of the glottis increased elasticity and rigidity.

To summarize this rather complicated situation, the final position assumed by the vocal cords in producing a tone, and the position in which it will undergo its vibrations, is the product of two muscular forces, one of which (the adductor mechanism) adducts the vocal cords and places them under varying degrees of tension and a second, a very finely regulated muscular mechanism (the pull of selected fibers of the thyroarytaenoidei), which counterpulls against the first over a varying length of the cord. This accounts, for example, for such phenomena as the variation in the lengths of cord approximated and separated in varying stages of the "damping" (or falsetto) mechanism, or in the degree of separation and tension of the entire length of the cords in the production of various tones of the lower range, which takes place in the so-called "normal" mechanics.[25]

Fig. 17.—Position of the vocal cords in the production of low guttural tones. The vocal cords are relatively relaxed, with the hiatus between them widest posteriorly.

DETAILS OF THE RELATIONSHIP OF CONDITIONS WITHIN THE LARYNX TO THE TONE PRODUCED

The appearance of the vocal cords as they produce a range of tones from the lowest to the highest can be described and illustrated with a considerable degree of accuracy. It is known that, beginning with the lowest possible tones any given larynx can produce, certain general changes in the position, length and tension of the vocal cords occur as the scale is ascended to the highest possible tone.[26]

The lowest tone any larynx can produce is of the "guttural" type not ordinarily used in speaking or singing. It is produced (fig. 17)

25. The concept just described, which is essentially one of pull and counterpull, has been discussed recently by Chevalier Jackson (Jackson, C.: Myasthenia Laryngis, Arch. Otolaryng. **32**:434 [Sept.] 1940) in an article published too recently to be given full consideration in this paper.

26. French.[2] Pressman.[18]

by a general relaxation of the vocal cords, which are foreshortened, with a relatively wide gap between them (not, however, so wide or so definitely triangular as in the inspiratory phase of deep respiration). This gap extends from the anterior commissure and is not of equal width throughout the length of the chink of the glottis; on the contrary, there is visible, beginning at about the level of the middle third and extending posteriorly, a further rather acute bowing outward, so that the posterior portion of the gap is considerably wider than the anterior portion. This foreshortening probably represents a sliding backward of the thyroid cartilage or a forward movement of the arytenoid cartilages, or both, which brings the two ends of the vocal cords closer together. In addition there is an approximation of the anterior portions of the cricoid and thyroid cartilages; thus, the cricothyroid gap is diminished. These movements likewise lessen tension of the vocal cord. Such combined action of foreshortening produces a "slack," which is recognized as the outward bowing of the cords themselves. It is accompanied by a certain degree of outward rotation and sliding apart of the arytenoid cartilages, which further separates the posterior segments. The false vocal cords similarly appear lax and more or less tend to approach the midline, partially obscuring the true cords. Vibrations of the cord margins are slow and wide. This mechanism, however, can be observed only in selected persons.

From this point on, as one ascends the scale, there is a gradual transition, consisting of a straightening of the cords and an increase in their tension. The posterior gap tends to be less noticeable as the cords elongate, tension becomes increased and the cord edges come to lie nearly parallel. The impression gained by observation of the action is one of a "drawing out" process. This is due to several factors. The cricoid becomes fixed in a more posterior position and the thyroid cartilage is drawn forward, so that the length between the extremities of the cords is increased. The cricothyroid gap is widened by a tilting upward of the thyroid cartilage, and the arytenoid cartilages slide toward each other on their facets, at the same time rotating inward toward the midline.

If it is assumed that the pressure of expired air is maintained at nearly constant levels, it is seen that further elevations in tone are accomplished by a continuation of the process described until the cords become almost exactly parallel (fig. 18) and further increases in tension and length take place (fig. 10), each step in the process producing a higher tone.[27]

27. Bell Telephone Company Laboratories,[22] quoted by Pressman, J. J.: Physiology of the Larynx, Laryngoscope **50**:86 (April) 1940.

However, at any point in this course of events, if volume is to be increased at the same time that the pitch is to be elevated, no change in the appearance of the larynx need take place in order that this higher tone may be produced, for the added pressure of escaping air is sufficient in itself not only to increase loudness but to elevate the pitch as well.[19]

THE DAMPING PROCESS

Finally a point is reached in the ascending scale at which the vocal cords are as taut and as elongated as possible, closely approximated, under extremes of tension and stimulated into vibrations by as powerful a blast of air as is compatible with the volume of sound desired. There then comes into action a new mechanism for further elevating the pitch, popularly known as the "falsetto," a term to which many objections can

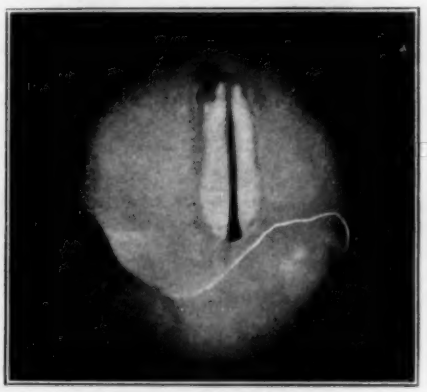

Fig. 18.—Position of the vocal cords in the production of middle tones. The enlarged posterior hiatus has disappeared, the margins of the vocal cords now paralleling one another. Variations in the length and tension of the cords and in their degree of approximation produce, with this same pattern, variations of pitch within reasonable limits.

be raised. I much prefer to identify this mechanism as the "damping process." This term accurately describes it, in keeping with known principles of the physics of sound production, with which principles it is closely identified.

A violin string in vibration throughout its entire length and not hampered by any fingering produces a given tone. If the string is then fingered so as to isolate functionally a portion of its length from the stimulation of the bow, the distal portion so isolated no longer vibrates and is said to be "damped." The remainder of the string, which is still free to vibrate, produces a higher tone, in keeping with the physical laws of vibrating strings. Such a phenomenon takes place in the larynx, by

means of which a segment of varying length of each vocal cord (always a continuous length occupying the more posterior portion of the cords) is damped and prevented from vibrating by the firm apposition of a portion of one cord to a corresponding portion of the other. The anterior segment which is free to vibrate is thereby functionally foreshortened, so that a higher tone is produced without the necessity for further increases in tension or the development of added expiratory pressure. This has already been discussed more completely in the para-

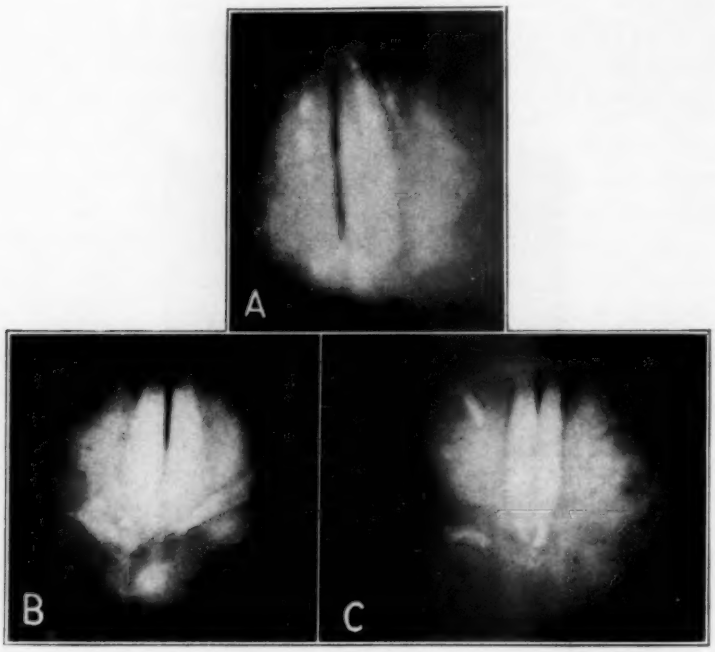

Fig. 19.—*A*, position of the vocal cords in the production of higher tones. First stage of the "dampening" (falsetto) mechanism. The posterior tips of the cords, by contact with one another, dampen the corresponding segment of the opposite cord, thus shortening the length of each cord which is free to vibrate. In general, with this position a higher tone would be produced than with that shown in figure 17, but, if under great tension, the cords in the latter position could produce the higher tone. *B*, position of the vocal cords approaching the production of the highest tones. The length of the dampened segments becomes increasingly greater and that of the segments free to vibrate therefore correspondingly shorter. The pitch is proportionately elevated. *C*, position of the vocal cords in the production of the highest tones. The dampened segments extend over almost the entire length of the vocal cords, leaving only a very small hiatus anteriorly. The segments free to vibrate are therefore very short, and the tone produced, extremely high. Further closure of the glottic chink would result in spastic aphonia, since no hiatus would be present for the escape of air.

graphs dealing with the theories concerning the mechanism of sound production at the larynx.

Damping of the vocal cords is accomplished by an appropriate segment of one vocal cord coming into direct and forceful contact with a corresponding segment of the other and the retention of this position for the duration of the production of the tone. The approximated segments of the cords have been damped, one by the other, and are prevented from vibrating, regardless of the pressure of the expiratory blast. Only the foreshortened portions of the cord anterior to the damped segments remain free to vibrate.

In passing from the mechanism of parallel cords to that of damped cords, the first tone or lowest tone of the damped series results from the approximation of just the posterior tips of the cords, thus leaving relatively long vibrating segments anteriorly (fig. 19 *A*). As higher

Fig. 20.—Laryngeal spasm from attempts to phonate at a pitch higher than this larynx can produce. The vocal cords are tightly approximated, the ary-epiglottic folds are being drawn toward the midline, and, despite the retraction of the epiglottis by the laryngoscope, the epiglottic tubercle is nevertheless closing in over the larynx. A later stage in the development of the spasm would demonstrate a typical sphincteric closure of the larynx, including approximation of the false vocal cords.

tones are produced in a gradually ascending scale, the length of segment damped by the other becomes longer and the vibrating portion correspondingly shorter (fig. 19 *B*). Finally, the cords approximate for so great a length that the chink of the glottis consists of nothing more than a tiny orifice located anteriorly (fig. 19 *C*).

The muscle action by means of which the length of the damped segment can be varied has been described in the paragraphs dealing with the action of the internal portion of the thyroarytaenoideus. In the main, it consists of the selective action of various groups of fibers of the

thyroarytaenoidei which hold apart, against tension, a varying length, beginning anteriorly, of each vocal cord, dependent on the tone to be produced (fig. 16). The length of cord thus bowed is equal on the two sides. Further damping beyond a point at which a tiny orifice is established cannot, of course, take place, since approximation beyond this point would find the cords completely closed, so that no expired air could pass, and a state of laryngeal spasm would ensue. When attempts are made to phonate at a pitch higher than the capabilities of the larynx permit, the resulting spasm consists of pronounced straining, generalized contraction of the soft tissues of the larynx under great tension, this process involving the false vocal cords and the aryepiglottic folds as well as the true cords (fig. 20), and elevation of the larynx toward the epiglottis and base of the tongue due to the action of the extrinsic muscles. The effort is ineffective and produces no satisfactory tone, usually terminating rapidly.

In the transition from the mechanism of parallel cords to damping there is sometimes noticeable a change in the quality of the voice, which transition is known as a "break."

ADDED PHENOMENA ACCOMPANYING THE PRODUCTION OF ANY TONE, REGARDLESS OF PITCH

Certain phenomena within the larynx are common to all tones, regardless of pitch. Laryngeal sounds take place, as I have already indicated, when the vocal cords are approximated by the action of the arytenoid cartilages and placed under tension by the action of the intrinsic and extrinsic musculature of the larynx. This is known as the "position of phonation." The cords, as they are brought into this position of complete adduction, seem on mirror and direct laryngoscopic observation to move from their lateral to their median positions as stiff rigid bands. Such, however, is not at all the case. The movement to the midline is rather an undulating one, in which the anterior segments seem to lead and the posterior segments to follow. Thus, the anteriormost portions come into closer approximation first, then the middle segments and finally the posterior segments. The movement is a smooth flowing one, recalling to mind the movement of a long flexible whip as it is gently flicked at some distant object. From the position of rest this preliminary undulating adductor movement is complete, bringing the vocal cords into practically total apposition and under tension which varies with the anticipated pitch of the tone to be produced (fig. 21). This phenomenon of a complete preliminary apposition apparently does not recur during the production of a tone or a series of tones unless a period of relaxation for a purpose such as respiration intervenes.

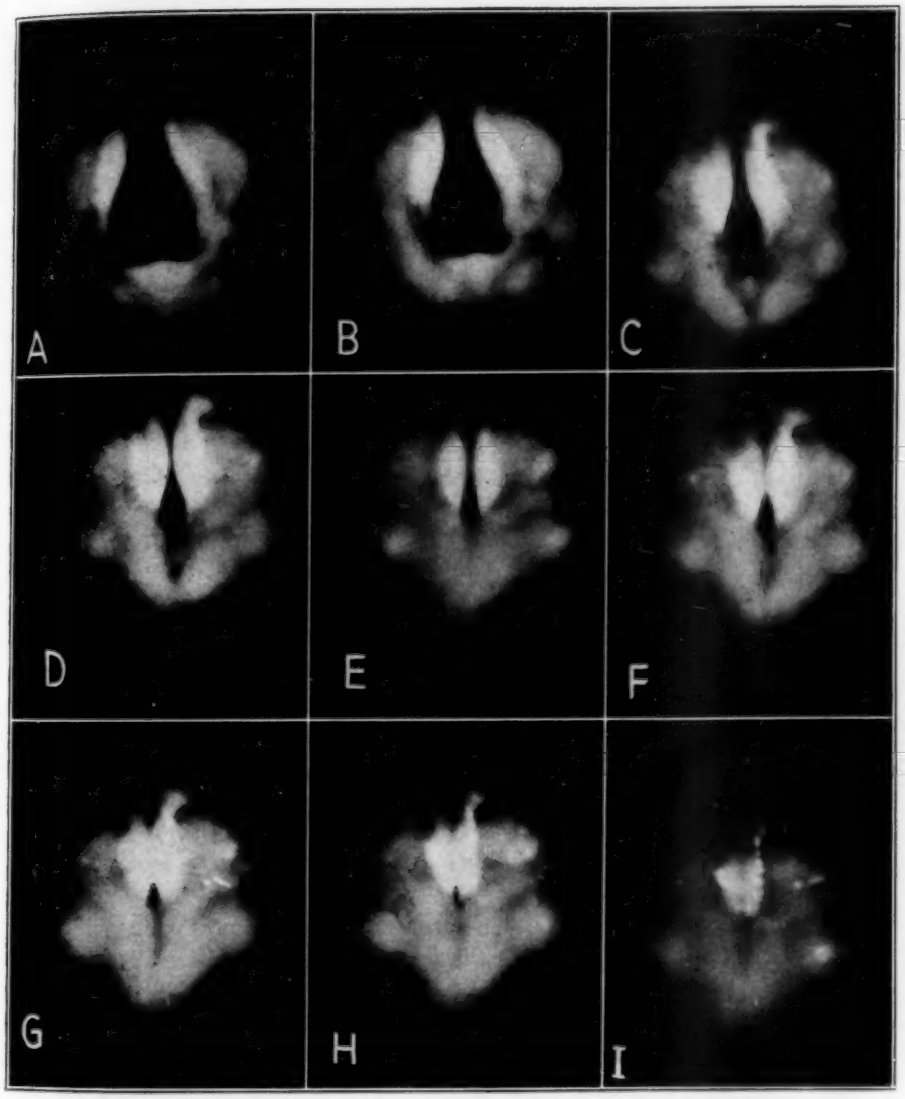

Fig. 21.—Photographs showing the undulating character of the movement which adducts and completely approximates the cords as a preliminary step to the setting up of vibrations which produce tone. Note how the anterior segments close first, to be followed by the more posterior segments. The final approximation of the cords is complete. It will be followed by a forceful separation of varying segments of the cords to the position of vibration, thus producing a variety of tones.

There are several possibilities as to the next, or vibratory, stage. It may well be that during the actual vibrations the cords enter into the separational phase of the vibrating position solely because of the force of the expired air and that therefore, so far as the vocal cords are concerned, this movement is entirely passive. Their active role, then, would manifest itself by a springing back into the more closely approximated position of the vibratory movement because of the factor of elasticity inherent in the tensed cords. This springing-back movement can occur against the pressure of expired air, because the pressure has been diminished somewhat as a result of the escape of a certain quantity of air at the instant the cords were blown apart. When the margins of the cords have been reapproximated as a result of this elastic recoil, pressure reaccumulates below them until a point is reached when this becomes sufficiently great to blow them apart again. It is, then, not unlikely that the vibrations of the cords result from the pressure of expired air and represent, as Guthrie and Milner [28] and others have pointed out, an aerodynamic phenomenon.

It is likely that the separation phenomenon involved in the vibratory cycle consists of two phases, which occur almost simultaneously. First, there is the distinct pulling apart of the cords by appropriate fibers of the thyroarytaenoidei acting against the combined pull of the adductors and against the inherent elasticity possessed by the cords themselves when under strong tension. This has already been likened to the backward pull of the taut bowstring by the arm of the archer, which latter represents the fibers of the thyroarytenoid muscle. The pull involves either the entire length of both cords or selective equal anterior segments of the two cords, depending on the pitch of the tone to be produced. When this outward pull has reestablished the chink of the glottis to the necessary length and width, in keeping with the pitch of the anticipated tone, air from below is forced through this chink, which in turn accounts for the second phase. In escaping, this air under pressure causes further separation, consisting of an outward and upward movement, not of the entire cord, but only of that portion which already has been separated by the muscular pull. This represents the beginning of the cycle of vibrations. Any portions of the margins of the vocal cords which are in complete approximation when this blast occurs remain approximated. The segments of the cord free to vibrate, having been blown apart into the separational phase of the vibratory movement, tend to spring back because of their own elasticity into the closed phase of the vibration. The pull of the thyroarytaenoidei, however, maintains whatever chink existed prior to the further blowing apart of the margins

28. Guthrie, D., and Milner, A., in Discussion on Functional Disorders of the Voice, J. Laryng. & Otol. **54**:261 (May) 1939.

by the air current, so that the closing phase of vibration does not represent a complete closure. These vibrations are entirely distinct from the abduction and adduction of the vocal cords caused by rotation of the arytenoid cartilages. The latter structures simply move the cords from the lateral position of respiration to the median position of phonation. When this has been done, the vibratory movements take place without any further movements of the arytenoid cartilages, the latter remaining absolutely fixed in their inwardly rotated position as the cords separate and approximate during their vibrations.

Regardless of what objections may be raised to the use of the term "falsetto" as denoting an entirely artificial distinction and giving the impression of a false, unreal or unnatural state of affairs, whereas the mechanism actually represents a perfectly normal logical step in the production of tones of a gradually ascending scale, the fact yet remains, regardless of the terminology, that such a configuration does take place. Those segments of the cords which are approximated under these conditions remain completely approximated throughout all phases of the vibratory cycle. Even during the production of the highest tones in a given singer's range, when he is forcibly straining to increase air pressure as much as is humanly possible, approximated segments of the cords remain tightly approximated, regardless of the force of the pressure exerted on them, and do not vibrate.

One sees, then, in summing up the situation, several stages in the production of any given tone:

1. The cords are adducted to the midline and placed under varying degrees of tension and elongation by the action of the adductor muscles.

2. Then either the entire length of the cords or varying segments of their more anterior portions, depending on the tone to be produced, are forcibly pulled apart, just a little, by the action of those internal fibers of the thyroarytaenoidei which insert into and become part of the cords. This is accomplished without any movement of the arytenoid cartilages, which remain tightly approximated.

3. The hiatus thus produced allows air to escape under pressure, which more or less everts the already separated free margins of the cords.

4. Of their own elasticity these everted edges of the vocal cords spring back into position without in any way affecting the degree of opening previously established by the pull of the thyroarytaenoidei, which remain as before.

5. The constant, very rapid repetition of this eversion and falling back of the margins of the vocal cords in a wavelike manner constitutes the vibrations of the cords.

DETAILS OF VOCAL CORD VIBRATIONS

Until recently little or nothing was known concerning the changes in contour or the type of movement taking place in the vocal cords during a single vibratory cycle; although stroboscopic observations had shed some light on the subject, they were not conclusive and led to wide variations in interpretation. Experimental observations by Hartmann and Wullstein [29] have, however, clarified the situation to some extent, so that it is possible to describe certain cross-sectional changes occurring in the cords as the vibratory movements take place. Essentially, these experiments consisted of allowing powerful points of light to shine through extirpated vocal cords which were forced to vibrate by air pressure from below and registering on a photoelectric cell the amount of light passing through the cords during the complete cycle of each vibration. As the cords thickened, less light passed through, and as they thinned, the amount of light reaching the photoelectric cell became greater.

The vibratory cycle is, in addition to the resting state, composed of two principal elements: an opening phase and a closing phase. The experiments of Hartmann and Wullstein described in the preceding paragraph, conducted on calf and human larynxes alike, seemed to prove that the cords are thickest and of the greatest bulk during the resting state. As vibrations are initiated and the closing phase begun, the cords become progressively thinned, reaching their maximum diminution in bulk just at the end of closure and as the opening phase is about to begin. This thinning just prior to the opening phase of the vibratory movements enables the cords to be blown apart more easily. As separation, or the opening phase, progresses, the cords thicken, but never to the degree that is present in rest. As the pitch is elevated, the cords are thinner during each comparable stage of the vibratory cycle.

The Bell Telephone Company's ultraslow motion pictures of the larynx, taken during phonation, at 4,000 frames a second and representing a slowing to 1:250 in the normal rate of vibration, demonstrate clearly the details of these vibratory movements. A descriptive folder is published by the Bell Telephone Company as an accompaniment to the film.[22] At low pitch the movements of the vocal cords are very complex, and they become less so as the pitch is elevated. The cords are relaxed, becoming firmer and elongated as the scale ascends. Beginning with the closed position of the cycle, it is apparent that they start opening from underneath, the opening progressing upward and outward. After they have opened, the closing phase similarly begins from underneath. As the folds open and close, a wavelike motion, or

29. Hartmann, W., and Wullstein, H.: Untersuchungen über den Bewegungsvorgang an den schwingenden Stimmlippin von Kehlkopfpräparaten mit verbesserter Photozellenmethode, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **144**:348, 1938.

ripple, passes laterally over the top surface. Also, at low pitch the folds may be nearly closed for half the vibratory cycle, and this relative period of closure becomes shorter in duration as the pitch is elevated.

At extremely high pitches only the edges of the cords nearest the glottis are seen to vibrate, so that variations in the width of the glottis during vibrations are very small, as contrasted with the considerable variation seen during the production of low tones. Furthermore, in support of previous descriptions outlined elsewhere, these films show that at a certain level of tone produced by the poorly named "falsetto mechanism" (I prefer the term "damping mechanism"), vibratory movements are, as the pitch is elevated, confined more and more to a diminishing anterior segment of the length of the cords, the more posterior segments remaining in firm contact one with the other.

The vibratory movements vary not only with the pitch but with the intensity of the sound produced. At very low intensities the cords close together feebly or may not close at all. At higher intensities but at the same pitch they close more firmly and may remain closed for an appreciable time. The widest opening during the vibratory cycle does not differ greatly with change in intensity. The amplitude of the vibratory movement therefore becomes greater as the pressure is increased, the added excursion representing a more complete movement to the midline. Curry, in his text,[6] has amplified this to the extent of pointing out that

> . . . increases in the air pressure above the minimal value necessary to initiate vibration at a given frequency determine the amplitude of the vibration and hence the intensity of the sound produced . . . following roughly the square law principle.

However, as I [30] have pointed out in a previous article, there is more to this problem than meets the eye. When volume is increased and vibratory excursions widened by virtue of added pressure, the latter factor of added pressure tends to elevate the pitch.[31] If, however, the pitch is to be maintained constant in the face of this added pressure, some compensatory change in the configuration of the cords must take place to lower the pitch in the same degree that the increase in pressure tends to elevate it. Thus, the cords would, for example, have to become less tense or some such equivalent change take place. Under circumstances, therefore, in which a pitch is to be maintained constant but volume is to be increased by added expiratory pressure, such increases in volume are accompanied not only by wider vibratory movement of the cords but also by changes to a pattern of vocal cord position

30. Pressman, J. J.: Physiology of the Larynx, Laryngoscope **50**:277 (April) 1940.

31. Negus,[19] p. 405.

characteristic of a relatively lower pitch. This change is required per se, as a compensatory measure against the tendency of the increased pressure to elevate the pitch. Pitch can thereby be maintained constant when desired, despite increases in volume. This would not be true if the larynx were simply a rigid orifice. In a peanut whistle, for instance, increases in intensity of sound are invariably accompanied by an elevation of pitch.

ACTION OF THE VENTRICULAR BANDS (FALSE VOCAL CORDS) IN PHONATION

The ventricular bands are in no way essential to the production of voice. In certain animals with very good voices, such as the cat, false vocal cords are entirely absent, or at least the true and false cords are blended into one.

Under normal circumstances the role played by the false cords in phonation is more or less a negative one. In many good singers the false cords appear to flatten out against the lateral wall of the larynx, thus diminishing their shelflike character. This opens the larynx above the vocal cords more widely and allows an uninterrupted passage at the level of the true vocal cords. In certain other persons the false vocal cords reverse this action during phonation and tend to approximate toward the midline. The resultant tone is comparatively unpleasant, probably on account of a muffling of the sounds produced and interference with the passage of sound waves from the larynx to the upper resonating chambers. This approximation also tends to reflect sound waves back down into the larynx. If the false cords are unusually large, they may come into direct apposition with each other, giving the voice a very husky quality. This approximation of the false cords, when it does occur, is not always present during the entire period of the production of a tone but may be transient, occupying only a portion of the cycle. Thus, for a momentary period the false cords may sweep across the larynx and then retract.

The chief action of the false cords in phonation appears to be that of the production of some of the overtones by a process of setting up secondary vibrations.

When the true vocal cords are in a markedly diseased state or if they have been removed surgically, the ventricular bands can be approximated and thus replace the true cords as phonatory organs. This is sometimes called "dysphonia plicae ventricularis."[32] The resultant voice is husky, rather low and with a small range of pitch. If false cord phonation in the presence of diseased true cords has been established

32. Jackson, C., and Jackson, C. L.: Dysphonia Plicae Ventricularis, Arch. Otolaryng. **21**:157 (Feb.) 1935.

for a long time, the mechanism may persist even after a healthy state of the true cords has been reestablished.

WHISPERING

The essential difference between whispering, on the one hand, and speech and singing, on the other, lies in the action of the arytenoid cartilages. In whispering, the sliding movement of the arytenoid on the cricoid cartilage to the midline, which approximates the medial wall of one arytenoid to that of the other, does not take place. This is due to failure of the interarytaenoidei muscles to contract. A wide triangular gap posteriorly is therefore always present during whispering (fig. 22). This posterior gap is bounded on its sides by the cartilaginous walls of the bodies of the arytenoids, which are nonvibrating, and the posterior

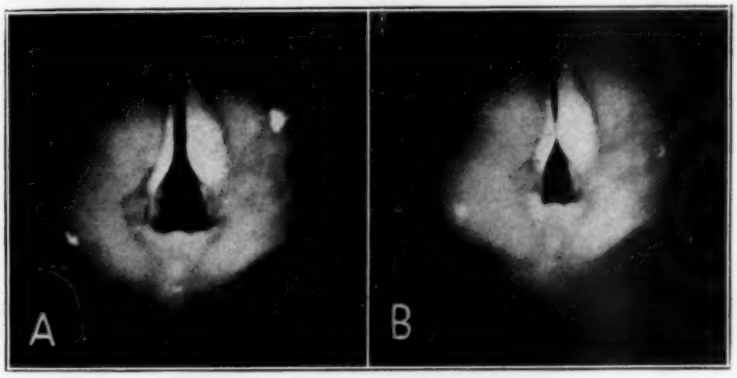

Fig. 22.—Position of the vocal cords in whispering. Note the very large posterior hiatus due to failure of the arytenoids to be approximated to the midline by the interarytaenoidei muscles. *A* represents a low-pitched whisper without effort and *B* an attempt to elevate the pitch and to whisper more forcefully.

segments of the vocal cords. Therefore, within the limits of this boundary a pronounced air leak can take place. The action of the anterior segments of the true vocal cords is not greatly unlike that found in speech. The anterior segments of the cords seem to vibrate to some extent at least, but there is no pronounced vibration in the region of the posterior gap. If an attempt is made to elevate the pitch of the whisper, the anterior oval hiatus may become narrower, the cords touch each other at about the middle third and the inward rotation of the arytenoids becomes a little greater, but the triangular chink found posteriorly continues to be present. This remains constantly open, and, since the cartilaginous walls do not vibrate, represents a large source of air waste, which gives the whisper its breathy, or aspirate, quality. A certain amount of contraction of the laryngeal sphincteric girdle and resultant partial approximation of the false vocal cords accompanies these movements.

LARYNGEAL MOVEMENTS DURING RESPIRATION

Since respiration forms so vital a part of phonation, laryngeal activities during breathing are appropriately a part of the study of phonation. The most obvious respiratory movements of the larynx consist of abduction of the cords away from the midline, with consequent widening of the chink during inspiration, and a relative adduction toward the midline, with narrowing of the chink during expiration. This movement is so slight in certain persons during quiet respiration as to be almost imperceptible, with the cords assuming, during rest, a position

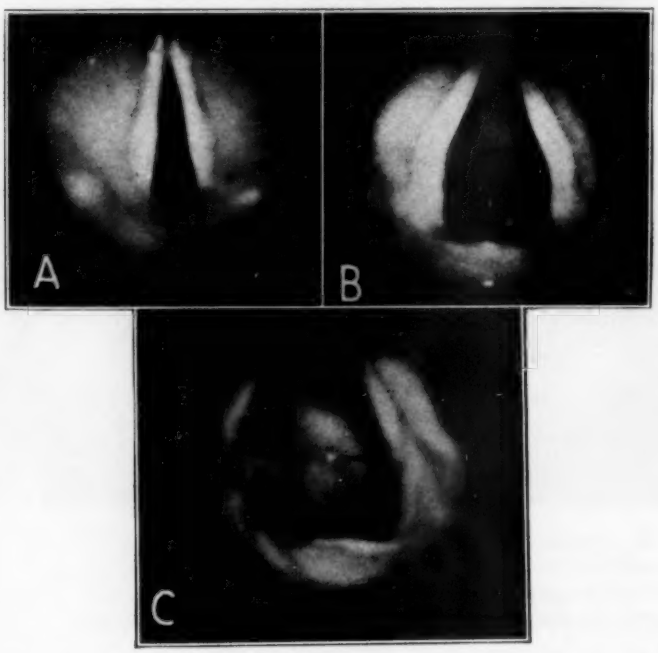

Fig. 23.—Position of the vocal cords during respiration. *A*, inspiration at rest. The expiratory phase cannot be distinguished easily from the inspiratory. *B*, relatively deep forced inspiration. The vocal cords are more widely separated than when at rest. During the expiratory phase the cords have essentially the same appearance as in *A*. In extremely deep forced inspiration (*C*) the vocal cords abduct widely to the lateral walls, offering almost no obstruction to the passage of air into the trachea, which is visible below.

just lateral to the midline (fig. 23 *A* and *B*). As the depth of respiratory movements increases, this excursion becomes easily recognizable. The lateral movement of the cords during inspiration becomes progressively wider as the inspiration becomes deeper and more forceful,[33] so that in

33. Canuyt, G.; Gunsett, A., and Greiner: La méthode des coupes radiographiques (tomographie ou planigraphie) appliquée à l'étude de la phonation. Rev. franç. de phonatrie **6**:133 (July) 1938. Pressman.[7]

very deep inspiration they may come to lie almost flush against the lateral walls of the thyroid cartilage, as a result of which the chink of the glottis becomes almost the same size as the lumen of the trachea (fig. 23 *C*). In this position they present practically no obstruction to the respiration and thereby, as, for example, in singing, permit the very rapid inhalation of large quantities of air.

During respiration, especially deep inspiration, it is necessary that the circular fibers of the cricopharyngeal pinchcock remain closed. These fibers are the lowest portion of the inferior constrictor of the pharynx and form a sphincter at the mouth of the esophagus. If this muscle band did not remain in a state of tonic contraction during respiration, more air would pass down the widely open mouth of the esophagus than into the relatively narrowed chink of the glottis.

Dessy [34] has described certain gross movements of the entire larynx which, in addition to movements of the vocal cords, take place during respiration. In inspiration there is generally a lowering of the larynx, and in about half the cases there is, in addition, a backward movement. In most people there is also a lateral deviation, always to the left. The amplitude of all these movements is very small. The downward movement comes about partially as a result of contraction of the appropriate ribbon muscles of the neck but also because of the lowering of the diaphragm and the increase of negative pressure within the thorax during the inspiratory phase. The backward movement and that to the left occur as an accompaniment of the downward movement, because the trachea in its downward course proceeds obliquely from front to back, receding from the surface as the deeper parts of the thorax are entered. The downward movement of the trachea is therefore necessarily accompanied with a backward movement of the trachea and the attached larynx. The lateral deviation of the larynx to the left during the inspiratory phase is believed due to the downward movement of the heart during inspiration and the traction which is thus exerted on the great vessels attached to the left main stem bronchus. In subjects who are breathing abdominally, these movements are even less pronounced.

NEUROGENIC CONTROL OF LARYNGEAL RESPIRATORY MOVEMENTS

The central nervous control of respiratory movements of the larynx is a dual one, in which medullary centers control involuntary respiratory movements and higher cortical centers control voluntary movements. Apparently, each of these two centers can, as the need arises, take over control of the other. For example, if one purposely takes a deep breath and holds it and brings the sphincter mechanism of the larynx into action to trap the air within the thorax, the resulting adduction of the vocal

34. Dessy, G.: Movimenti respiratori del laringe in condizioni normali e patologiche, Riv. di pat. e clin. d. tuberc. **12**:791 (Nov. 30) 1938.

cords is entirely a conscious effort controlled by cortical centers, just as the control of the cords in phonation is a voluntary cortical effort. It is possible, if one so desires, to maintain this position of the cords for a limited period or to abduct the cords abruptly and inspire at will. In this sequence of events the cortical centers control the medullary centers. However, after the breath has been held for a sufficiently long period and the accumulation of carbon dioxide in the blood stream has reached a sufficiently high level, the resulting stimulation of the respiratory centers in the medulla becomes so powerful that it is impossible to hold the breath any longer and the cords abduct as part of a forced involuntary inspiration.[24] In this situation, then, the lower, or medullary, centers have taken over control of the higher cortical centers.

These subcortical centers can control the respiratory movements of the larynx in a quantitative way. If in rarefied air or under pathologic conditions or during exertion the respiratory requirements are such that exaggerated respiratory movements are necessary, the vocal cords abduct more widely than during quiet respiration, to permit the passage of larger quantities of air in a short interval of time (fig. 23 *B* and *C*).

This dual control mechanism can be seen to function in a great many other circumstances. Under anesthesia, or during sleep, for example, respiratory laryngeal movements continue to take place entirely as the result of control by the subcortical centers, without intervention of any conscious control, and yet when the cortex is able to function such intervention can take place.

The cortical center controlling the voluntary respiratory movements of the larynx is probably located "in the frontal lobe in front of the motor area for trunk movements" [35] and, according to the Jacksons,[36] "in the insular or preinsular convolutions near Broca's area." The medullary centers controlling involuntary movements are presumably two in number, one for respiration and another for expiration. These are located in the medulla, near the nucleus ambiguus of the vagus nerve, and from them originate those involuntary impulses which stimulate the nucleus ambiguus into activity. From this nucleus fibers arise which later become the recurrent laryngeal trunks of the vagus nerve. In the recurrent trunk, fibers to the abductor muscles are supposed to travel in a separate bundle from those of the adductor group (although King [37] presented the opposite view), giving rise to the impression that there are separate and distinctive groups of cells in the nucleus ambiguus, one to control dilation and the other to control the adductor muscles. It has been suggested that at the end of inspiration impulses passing up

35. Mott, F. W., cited by Negus.[19]

36. Jackson, C., and Jackson, C. L.: The Larynx and Its Diseases, Philadelphia, W. B. Saunders Company, 1937, p. 24.

37. King, B. T.: Bilateral Abductor Cord Paralysis, Northwest Med. **38**:125 (April) 1939.

the vagus nerve stimulate the center for expiration and that at the end of expiration a second group of impulses stimulates the center for inspiration. This is known as the Hering-Breuer reflex. In the final analysis, however, these reflex movements are controlled by the carbon dioxide content of the blood, which determines not only the rate but also the depth of respiratory movements, and most reflexes concerned with respiration are probably secondary to this blood carbon dioxide level.

PRACTICAL APPLICATIONS OF THE PHYSIOLOGY OF THE LARYNX

While most laryngeal diseases can perhaps be treated as well by a laryngologist whose knowledge of laryngeal physiology is less than it might be, an attempt to clarify the fundamentals needs no defense. And yet, if it were required to demonstrate that such fundamental knowledge would be worth while only if it could be applied in a practical way, such an association could be demonstrated easily. Whispering is a case in point. Many pathologic and postoperative convalescent states are treated by vocal rest. The advisability of permitting a patient to whisper instead of speaking requires, on the part of the laryngologist, an intimate knowledge of what happens in the larynx during whispering. To those familiar with the physiology involved, it is at once apparent that whispering can be as harmful as shouting, for the vocal cords can approximate and traumatize each other during the whisper as well as in speech, not to mention the fact that the expiratory blasts themselves produce vibrations of the vocal cords and consequent trauma. Without such knowledge, advice on this point is simply guesswork, and the laryngologist must always remain in doubt as to the wisdom of his decision on the issue. The evidence on which he deprives a patient of whispered oral communication is not founded on his knowledge of the reason for forcing this hardship on a patient, and yet if, as a guess, whispering were to be permitted, great harm might result. There can, however, be no doubt or guesswork in the mind of a laryngologist who understands the physiology of whispering.

Even more to the point is an example I recently encountered. The patient, a professional singer, enjoying a large income from the practice of his art, gradually became hoarse and consulted a laryngologist, who ultimately removed, with great skill, a small benign neoplasm from the margin of the posterior third of one vocal cord. After healing had taken place, the patient resumed his singing and the hoarseness promptly recurred. He also noticed from the beginning of resumption of singing that high tones were produced only with effort. His voice frequently "broke" in the higher ranges, and it had a peculiar quality which he described as an "air leak." More recently hoarseness had been present not only when high tones were produced but throughout the scale. His laryngologist found the vocal cords to be injected and somewhat edema-

tous, with a small concavity at the site of the tumor, but rightfully assured the patient that there had been no recurrence of the growth. After a period of vocal rest and a second resumption of singing the same cycle again came to pass, so that this artist was forced to discontinue his career, which had been outstanding. What had happened was this: A small depression had resulted, probably from contraction of scar tissue at the tumor site. This vocalist, with a large range of tones, had always and still did produce the higher notes in the scale by the damping (or "falsetto") mechanism, so that the posterior thirds of the cords were approximated over the area of the postoperative concavity during the production of these higher tones. The concavity in the margin of one cord had, therefore, allowed an air leak during approximation, as well as production of secondary tones throughout the higher range. The patient's trained ear recognized this, and in a conscious or subconscious attempt to close this leak he more forcefully approximated the cords, resulting in frequent occurrence of a laryngeal spasm which he described as a "break in his voice." There was always, therefore, a tremendous degree of laryngeal straining in a hopeless effort to close the leak, and a resultant traumatic laryngitis ensued, which was the cause of his hoarseness on each attempt to resume singing. No local therapy, no period of vocal rest—nothing, in fact, except the practical application of the fundamentals of laryngeal physiology—could have restored this patient's voice and permitted him to resume his career. In consultation with his vocal teacher, the larynx was observed during the production of the tones of an ascending scale. When that tone was reached for which the approximation of the posterior segments was required and the presence of the small pathologic hiatus became noticeable, the vocal teacher was so advised. The singing of tones at and above this level was forbidden. Tones produced below this level were not affected by the small concavity in the margin of the vocal cord, since no secondary air leak could occur with the cords physiologically separated at this point. No laryngeal straining was required, and no further inflammatory changes occurred. By the simple expedient, therefore, of placing the singer's range a few tones lower, which, with a short period of training was easily accomplished, he was enabled to resume his career, which today is as successful as before. This happy result is, then, just one of many examples in which only the application of the fundamentals of laryngeal physiology and nothing else could possibly have produced a clinical cure. Such examples are, however, totally unnecessary. Discussions of fundamental basic principles require no justification, and the knowledge of such things is, like virtue, its own reward.

Mr. Arthur Hinman gave technical assistance in obtaining the laryngeal motion pictures from which many of the photographs in this paper have been made.

1917 Wilshire Boulevard.

LOCAL USE OF SULFATHIAZOLE IN MANAGEMENT OF SIMPLE MASTOIDECTOMY WOUNDS

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Within the past few years there has accumulated a voluminous literature concerning the oral and parenteral administration of sulfanilamide and its derivatives, but the local use of these drugs has received little prominence. The excellent results obtained by Jensen and his associates [1] in the treatment of compound fractures with powdered sulfanilamide have served to arouse considerable interest in this type of therapy, and the work of Thomson [2] as well as that of Key and his co-workers [3] along similar lines has tended to place such therapy on a firm footing. Sulfanilamide has been found to be of value also in the local treatment of gonorrheal ophthalmia [4] and of chancroid.[5] Mueller,[6] in a recent communication to *The Journal of the American Medical Association,* described the local employment of this drug in cases of peritonitis with gratifying results.

The rapid healing of purulent, sloughing mastoidectomy wounds after the local application of sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole) crystals suggested to us the possibility of using this drug postoperatively in all cases of acute mastoiditis. It seemed reasonable to expect that such therapy might lessen complications, shorten hospitalization, decrease drainage and in general smooth the postoperative course. This study, therefore, represents an effort to evaluate the worth of such a method.

From the Manhattan Eye, Ear and Throat Hospital.

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It is based on a series of unselected consecutive cases in which operation was performed at the Manhattan Eye, Ear and Throat Hospital by us during the period from January through April 1941. In 20 cases used as controls the wound was treated in the routine manner, while in an equal number the wound was packed with sulfathiazole gauze at the time of operation. All the patients were seen during the same time of year, operated on with the same technic and similarly treated postoperatively except for the local use of sulfathiazole. None received sulfanilamide or any of its derivatives orally or parenterally before or after operation.

PROCEDURE

Complete simple mastoidectomy was carried out in the classic manner, care being taken in each instance to remove all of the cells. As soon as the mastoidectomy cavity had been completely exenterated, it was flushed with warm physiologic solution of sodium chloride, to remove any remaining debris. In the control series the cavity was packed with half-inch plain or iodoform gauze packing and the wound closed with clips. No periosteal or mattress sutures were used. The external canal was packed tightly with plain gauze, which was not disturbed for five to six days. In the series in which chemotherapy was used, a similar procedure was followed in every respect except that the plain gauze packing was steeped in a paste made by mixing sulfathiazole crystals (2 to 3 Gm.) with sufficient sterile water. The sulfathiazole was not sterilized but was poured directly from its original container into a sterile medicine glass. Wound clips were taken out on the third or fourth day, and beginning on the second or third day a few centimeters of the packing gauze was withdrawn daily, until by the sixth or seventh postoperative day all had been removed. Thereafter, the lower limit of the wound was kept patent with a Sirus drain until cessation of drainage permitted removal of the drain.

ACTION OF THE DRUG

The action of sulfathiazole, as the drug was employed in these cases, appears to be exerted solely at the site of use and not through the blood stream. Absorption through bone is slow; the absorption which takes place occurs almost entirely through the soft tissues. In at least one third of the cases the level of sulfathiazole in the blood was determined the day after the operation. In most of them none of the drug was present in the circulating blood, while in a few, a trace was found, the highest determination being 0.7 mg. per hundred cubic centimeters. This proves conclusively that the concentration in the blood is not sufficient to be therapeutically active. Because the concentration in the blood is negligible, the untoward reactions encountered at times, when the drug is employed systemically, need not be feared.

Since the action of sulfanilamide and its derivatives, both in vitro and in vivo, has been found to be bacteriostatic rather than bactericidal, it seems logical to assume that when the drug is used locally its action is exerted along similar lines. However, the high local concentration of the

drug which can be brought to bear on an offending organism must account for the drug's effectiveness in such instances.

Sulfanilamide and its derivatives do not exert any deleterious effect on the tissues with which they come in contact. Key and his colleagues, in their work with dogs, were able to demonstrate that when powdered sulfanilamide was placed in wounds it was well tolerated. They expressed the belief that perhaps healing of wounds was retarded slightly, but they were unable to prove this experimentally.

Sulfathiazole has been found to be the drug of choice in dealing with Pneumococcus and Staphylococcus. It is practically as effective as sulfanilamide in controlling streptococcic infections. For this reason sulfathiazole was employed in the cases used for our study, and no attempt was made to vary the drug with the organism obtained by culture of material removed at myringotomy. On account of the sparse solubility of the drug, the crystals were not packed into the mastoid cavity, since it was feared that a foreign body reaction might set in.

ANALYSIS OF DATA

In tables 1 and 3 the pertinent facts concerning the untreated patients are listed, while similar data with regard to the sulfathiazole-treated patients are recorded in tables 2 and 4.

Age.—The youngest patient in each group was 1 year of age, while the oldest was 58. The great majority of the patients were children, but 3 adults are included in the control series and 2 in the treated series. Age apparently is not a factor to be considered, since in the older patients the course was essentially the same as in the younger ones.

Predisposing Diseases.—In practically all the cases acute mastoiditis appeared as a sequela of measles, the common cold, influenza, whooping cough or mumps. In the control series acute mastoiditis followed measles in 4 cases (20 per cent) and the common cold in 12 cases (60 per cent). Influenza and postoperative tonsillectomy and adenoidectomy accounted for 1 case each. In 2 cases there was no history of a preceding illness.

In 7 cases (35 per cent) of the series in which sulfathiazole was used, the mastoiditis followed measles and in 7 cases (35 per cent), the common cold, while in 3 cases it occurred after the following diseases, respectively: mumps, influenza and pertussis. In this series 3 patients had not suffered from a preceding disease.

Roentgen Findings.—Roentgen evidence of acute mastoiditis was present in every case.[7] Four plus involvement of the mastoid cells,

7. Three plus involvement indicates a marked degree of opacity and obliteration of cell walls; incipient softening of slight degree may be present. In four plus involvement actual softening and absorption are present frequently and abscess formation as indicated by coalescence due to breakdown of all walls.

usually with softening or absorption, or with both, was reported in all but 1 case—that of a sulfathiazole-treated patient who showed 3 plus involvement without absorption or softening.

TABLE 1.—*Control Series*

Case	Sex	Age, Yr.	Sequela of	Duration	Pathologic Changes	Organism
1	F	2	Measles	3 weeks	Pus and granulations	Str. haemolyticus
2	M	58	Cold	6 weeks	Pus and granulations	Str. haemolyticus
3	F	7	Cold	2 weeks	Subperiosteal abscess	Friedländer bacillus
4	F	4	Postoperative tonsillectomy and adenoidectomy	7 weeks	Pus and granulations	Str. haemolyticus
5	F	17	.....	14 weeks	Pus and necrosis	Str. haemolyticus
6	M	54	Cold	1 week	Granulations	Unclassified pneumococcus
7	M	39	Cold	5 weeks	Granulations	Str. haemolyticus
8	F	5	Cold	3 weeks	Subperiosteal abscess	Staph. aureus
9	M	6	Influenza	3 weeks	Exudate and pus	Culture misplaced
10	M	9	Cold	1 week	Subperiosteal abscess	Staph. aureus
11	F	1¼	Cold	4 weeks	Subperiosteal abscess	Staph. aureus
12	F	8	.....	1½ weeks	Pus	Str. haemolyticus
13	F	1⅔	Cold	1 week	Subperiosteal abscess	Str. haemolyticus
14	M	2	Cold	3½ weeks	Granulations	Staph. aureus
15	F	7	Measles	3 weeks	Subperiosteal abscess	Str. haemolyticus
16	F	2	Cold	5 weeks	Pus and granulations	Str. haemolyticus
17	M	3½	Cold	3 weeks	Pus and granulations	Str. haemolyticus
18	F	10	Measles	8 weeks	Subperiosteal abscess	Pneumococcus type III
19	F	1	Measles	3 weeks	Pus and granulations	Str. haemolyticus
20	M	5	Cold	2 weeks	Granulations	Str. haemolyticus

TABLE 2.—*Series in Which Sulfathiazole Was Used*

Case	Sex	Age, Yr.	Sequela of	Duration	Pathologic Changes	Organism
1	M	7	Cold	5 days	Granulations	Str. haemolyticus
2	F	5	Measles	5 weeks	Pus and granulations	Str. haemolyticus
3	M	2½	Cold	4½ weeks	Pus and granulations	Str. haemolyticus
4	M	5	Mumps	5 weeks	Subperiosteal abscess	Staph. aureus
5	M	6½	Cold	3 weeks	Granulations	Staph. aureus
6	M	54	.....	2 weeks	Necrosis	Pneumococcus type III
7	M	2½	.....	3 weeks	Pus	Str. haemolyticus
8	M	1	Cold	2 weeks	Pus and granulations	Str. haemolyticus
9	M	5	Measles	5 weeks	Pus and granulations	Staph. aureus
10	M	11	Cold	2 weeks	Pus and granulations	Staph. albus
11	F	5	Measles	3 weeks	Granulations	Staph. aureus
12	M	43	Influenza	5 weeks	Pus and granulations	Staph. albus
13	F	7	Measles	3½ weeks	Subperiosteal abscess	Str. haemolyticus
14	F	6	Measles	3 weeks	Pus and granulations	Str. haemolyticus
15	F	7	Whooping cough	5 days	Subperiosteal abscess	Staph. aureus
16	M	16	.....	6 days	Subperiosteal abscess	Str. haemolyticus
17	F	2	Cold	2 weeks	Pus and granulations	Str. haemolyticus
18	F	2	Measles	5 weeks	Pus and granulations	Str. haemolyticus
19	F	17	Cold	1 week	Subperiosteal abscess	Pneumococcus type III
20	M	5	Measles	2 weeks	Granulations	Staph. aureus

Pathologic Changes.—At operation all the patients revealed definite cellular destruction with free pus and granulations. Subperiosteal abscesses were found in 7 of the untreated patients (35 per cent) and in 5 of the treated ones (25 per cent).

Bacteriologic Observations.—The causative organism in acute mastoiditis has been repeatedly found to vary from year to year and from season to season. In this series perhaps the most surprising finding is the

TABLE 3.—*Control Series*

Case	Hospitalization, Days	Postoperative Drainage, Days	Complications
1	24	34	Disruption of wound
2	18	25	None
3	17	27	Cellulitis of wound
4	19	32	None
5	13	19	None
6	14	34	None
7	10	17	None
8	32	41	Disruption of wound twice
9	12	24	None
10	10	18	None
11	39	51	None
12	17	35	None
13	13	36	Postauricular abscess
14	8	18	Measles
15	10	20	None
16	52	52	Disruption of wound twice
17	12	19	None
18	19	28	None
19	36	36	Still draining
20	43	66	Measles
Average	24.9	31.6	

TABLE 4.—*Series in Which Sulfathiazole Was Used*

Case	Hospitalization, Days	Postoperative Drainage, Days	Complications
1	20	21	None
2	8	18	Postauricular abscess
3	10	14	None
4	12	12	None
5	13	15	None
6	9	16	None
7	6	9	None
8	12	17	Measles and otitis media purulenta acuta
9	12	15	None
10	8	11	None
11	7	17	None
12	6	10	None
13	8	10	None
14	9	14	None
15	9	14	None
16	8	19	None
17	6	13	None
18	7	18	None
19	8	12	None
20	11	23	None
Average	9.4	14.9	

large number of cases in which Staphylococcus was the etiologic agent. The possibility that the cultures represent contaminants has practically been eliminated, since all the material for culture was taken directly from

the mastoid cavity and sent immediately to the laboratory. The bacteriologic observations are summarized in table 5.

Hospitalization and Drainage.—Comparing the postoperative healing time in the two series, one notes that recovery was more rapid in the patients treated with sulfathiazole. In this group the length of hospitalization ranged from six to twenty days, while the postoperative drainage varied from nine to twenty-three days. The hospital stay of the control patients lasted from eight to fifty-two days, with postoperative discharge continuing from seventeen to sixty-six days. The average drainage time of the control series of wounds was thirty-one and six-tenths days, as compared with fourteen and nine-tenths days for the sulfathiazole-treated ones. These statistics indicate that among the patients treated locally with sulfathiazole the average time required for healing was shortened by sixteen and seven-tenths days or reduced by slightly more than one half.

TABLE 5.—*Summary of Bacteriologic Observations*

	Patients Treated with Sulfathiazole	Untreated Patients	Total Percentage
Streptococcus haemolyticus	10	12	55
Staphylococcus	8	4	30
Pneumococcus type III	2	1	7.5
Friedländer bacillus	..	1	2.5
Pneumococcus (unclassified)	..	1	2.5
Culture misplaced	..	1	2.5

REPORT OF CASES

Cases 1 and 2 are those of children, sisters, one of whom was treated with sulfathiazole locally while the other received no chemotherapy. These patients are of particular interest because of the similarity of age, disease process and causal organism. The rapid healing of the treated wound as contrasted with the progress of the untreated one is striking.

CASE 1 (case 8 in tables 1 and 3).—A white girl 5 years of age was admitted to the Manhattan Eye, Ear and Throat Hospital with a history of purulent otorrhea on the left side, of three weeks' duration, which followed a cold. The report of the roentgen examination of the mastoid process was 4 plus involvement with marked absorption. A subperiosteal abscess was present, and culture of material taken at the time of mastoidectomy revealed Staphylococcus aureus. The wound was packed with iodoform gauze and closed with clips. After one week the wound had broken down completely, necessitating plastic closure. Again the wound broke down, and plastic closure was once more carried out. Permanent healing was accomplished forty-one days after the operation, with a hospital stay of thirty-two days.

CASE 2 (case 15 in tables 2 and 4).—A white girl aged 7 years, the sister of the patient in case 1, presents a striking contrast so far as her postoperative course is

concerned. She was admitted to the Manhattan Eye, Ear and Throat Hospital less than two months after her sister, with purulent otorrhea on the left side, of five days' duration, a sequela of pertussis. She, too, had a subperiosteal abscess, and the organism cultured from the mastoid cavity was also Staph. aureus. Sulfathiazole gauze was used to pack the wound. In nine days the patient was discharged from the hospital, and in fourteen days the wound was completely healed.

The effect of sulfathiazole applied locally in accelerating healing is illustrated in cases 3, 4 and 5. Bilateral simple mastoidectomy was performed; the cavity on one side was packed with the chemotherapeutic agent while that on the other side was used as a control.

CASE 3.—A white boy aged 4 years was admitted to the Manhattan Eye, Ear and Throat Hospital with a history of bilateral aural discharge of two weeks' duration, a sequela of measles. When simple mastoidectomy was carried out on the left side, the cells were found to be necrotic and a culture was reported to yield hemolytic Staph. aureus. The cavity was packed with iodoform gauze. Discharge continued for thirty-one days, at which time healing was complete.

Two weeks after the first operation a simple mastoidectomy was performed on the right side. The pathologic and cultural findings were identical with those of the opposite side. The wound was packed with sulfathiazole gauze. No aural discharge occurred at any time following the operation, and by the sixteenth postoperative day the wound was completely healed.

CASE 4.—A 20 month old Negro infant was admitted to the Manhattan Eye, Ear and Throat Hospital with a history of otorrhea on the left side of five weeks' duration. A roentgen examination of the mastoid process was reported to show 4 plus involvement with absorption. Simple mastoidectomy was carried out and the cavity packed with sulfathiazole gauze. Streptococcus haemolyticus was cultured from the wound. Healing was completed by the twenty-first day.

Ten days after the simple mastoidectomy on the left side, the right ear began to drain. Five days thereafter the patient began to have a fever, the temperature varying between 100 and 103 F., with evidence of mastoid involvement. The next day simple mastoidectomy was performed on the right side, the cavity being packed with plain gauze. Hemolytic Staph. aureus was obtained by culture. After the operation the wound discharged profusely, but the patient's course was uneventful until the twelfth postoperative day, when a pronounced septic fever began. A blood culture was positive for a nonhemolytic strain of Streptococcus. The sigmoid sinus was explored, but the sinus appeared normal and no clot could be demonstrated. The sinus was packed with iodoform gauze and the wound left open. Three weeks after the last operation drainage is still continuing.

CASE 5 (case 20 in tables 1, 2, 3 and 4).—A 5 year old white boy was admitted to the Manhattan Eye, Ear and Throat Hospital with a discharging left ear of two weeks' duration. Roentgen examination showed a 4 plus involvement of the mastoid cells, with moderate softening. Simple mastoidectomy was performed on the left side, and the mastoid cells were found to be filled with granulations and free pus. Culture of material taken from the mastoid cells at the time of operation yielded Str. haemolyticus. The cavity was packed with plain gauze and the incision closed with clips. Six weeks after the operation, while the patient was still in the hospital, measles developed. He was transferred to the Willard Parker Hospital, where the wound continued to drain for another three weeks. The total time during which the wound drained was sixty-six days.

During the attack of measles, acute purulent otitis media and mastoiditis developed on the opposite side, and a little over two months after the first operation simple mastoidectomy was carried out on that side. The pathologic changes were similar in every respect to those seen in the other mastoid. Culture of material taken at operation yielded Staph. aureus. On the eleventh postoperative day the patient was discharged. The wound drained for a total of twenty-one days.

Cases 6 and 7 represent the only complications occurring in the series in which the sulfathiazole treatment was used.

CASE 6 (case 2 in tables 2 and 4).—A 5 year old white girl had noticed purulent aural discharge from her right ear for five weeks following an attack of measles. Evidence of 4 plus involvement of the affected side, with softening, was reported observed in the roentgenogram. At operation the mastoid cells were found to be filled with pus and granulations, and considerable breaking down had occurred. Str. haemolyticus was cultured from the mastoid wound. The cavity was packed with sulfathiazole gauze. On the sixth postoperative day the patient was discharged from the hospital with a dry middle ear, and by the eighteenth postoperative day the postauricular wound had completely healed. Ten days later a postauricular abscess developed, which promptly healed after simple incision and drainage.

CASE 7 (case 8 in tables 2 and 4).—A 1 year old white boy was admitted to the Manhattan Eye, Ear and Throat Hospital with a purulent discharge from the left ear of two weeks' duration, which developed after acute coryza. Roentgen examination showed 4 plus involvement with absorption. Granulations and free pus were found at operation, the causative organism being Str. haemolyticus. The wound was packed with sulfathiazole gauze. At the end of seventeen days complete healing had taken place. One week later measles developed, and acute purulent otitis media on the left side occurred as an aftermath. Drainage from the canal ensued for ten days.

COMMENT

Although the number of cases in this series is small, the marked decrease in both the postoperative hospitalization time and the drainage time as well as the great reduction of complications in the series in which chemotherapy was used appears significant. In addition, it was found that with the treated patients discharge from the middle ear ceased almost immediately after operation while with many of the untreated ones aural discharge continued for some time.

While purulent discharge, usually profuse, occurred in practically all of the untreated wounds, a thin, slightly blood-tinged serous discharge appeared in the treated wounds and rapidly diminished until complete cessation resulted. In many instances this difference was so marked that the treated wounds could be distinguished from the untreated ones merely by examining the external dressings.

Healing was found to be accelerated rather than retarded in the sulfathiazole-treated wounds, but since in all the cases with which we have dealt the wounds were infected and the drug served to control the infection and thus to promote healing, this observation may be erroneous. Bleeding was found to occur frequently when dressings were

changed and the packing shortened. In no instance was this profuse or even troublesome. The growth of healthy granulations into the meshes of the gauze takes place fairly rapidly when sulfathiazole is used, and when these granulations are disturbed by redressing or by removal of packing, bleeding ensues. This explanation may account for the oozing seen at such times.

Recently, we have used sulfathiazole after modified and radical mastoidectomy. The method employed in these circumstances is the insufflation of the crystals into the mastoid cavity through the external auditory meatus. Sufficient data has not accumulated to permit of an accurate evaluation, but one gains the impression that the postoperative discharge is markedly decreased, and the odor frequently present in these conditions is almost entirely eliminated.

SUMMARY

Sulfathiazole was employed locally after simple mastoidectomy in 20 unselected cases, while in 20 other unselected cases serving as controls the wound was treated in a similar fashion except for the omission of sulfathiazole. In the series of cases in which chemotherapy was used, the drainage period averaged sixteen and seven-tenths days, or 52.8 per cent less than that in the control series, while postoperative hospitalization was reduced an average of eleven and four-tenths days, or 55 per cent. Reports of cases illustrating the value of sulfathiazole used locally in simple mastoidectomy wounds are included, as are reports of 2 cases in which complications occurred in the sulfathiazole-treated patients.

We believe that healing is promoted with minimum suppuration by the use of sulfathiazole in simple mastoidectomy wounds.

Drs. Marvin F. Jones, John R. Page, James G. Dwyer, Francis W. White, Daniel S. Cunning, Fred W. Graef, Robert E. Buckley and Joseph D. Kelly gave permission for the use of cases from their clinics.

210 East Sixty-Fourth Street.

IMPORTANCE OF CONTROL OF A PULMONARY LESION WHEN TUBERCULOUS TRACHEOBRONCHITIS COEXISTS

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The present extended use of bronchoscopy for the detection of lesions of the larger airways in tuberculosis has finally enabled the clinician to recognize the importance of tuberculous tracheobronchitis. This is shown clearly in the volume of case reports and papers which have accumulated in the literature, particularly during the past five years. As experience has enlarged, the pathogenic course, the signs and symptoms, and the associated roentgenographic pulmonary changes of tuberculous tracheobronchitis have become familiar to clinicians. Divergence of opinion occurs, however, concerning the advisability of pulmonary collapse and the necessity or value of certain local measures utilized in the treatment of the tracheobronchial lesions. It seems that the general management in cases of this type will remain a controversial subject until more endoscopic, clinical and laboratory findings are analyzed and correlated. Samson,[1] Samson and co-workers,[2] Myerson,[3] Barnwell, Littig and Culp,[4] Kernan,[5] McIndoe and associates,[6] Hawkins,[7] Peirce and Curtz-

From the departments of medicine and otolaryngology, Hudson County Tuberculosis Hospital.

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(Footnotes continued on next page)

wiler,[8] Warren, Hammond and Tuttle[9] and others have contributed materially to the knowledge of and current opinions on the problem, and it is superfluous to supplement the literature further unless one can present additional significant findings.

This report is a joint contribution of bronchoscopists and clinicians and represents their experiences in 302 bronchoscopic examinations of 137 selected patients with pulmonary tuberculosis, in 79 (58 per cent) of whom the complication of tracheobronchitis was present. The percentage of cases in which the presence of clinically suspected tuberculous tracheobronchial disease has been confirmed bronchoscopically has risen from 26, in the 1937-1938 period of the study, to 69, in the 1940-1941 period. In all the cases of this series bronchoscopic examination was

TABLE 1.—*Division of Cases at Time of Bronchoscopic Examination*

Group	Pulmonary Lesion *	No. of Patients	
		With Tracheo-bronchitis	Without Tracheo-bronchitis
A	Without demonstrable cavity	55	30
B	With demonstrable cavity or evidence suggestive of cavity	24	28

* Infiltration, bronchiectasis, atelectasis.

TABLE 2.—*Incidence of Symptoms**

Group	With Symptoms	Without Symptoms
With tracheobronchitis	65 (82%)	14 (18%)
Without tracheobronchitis	33 (57%)	25 (43%)

*Chief suggestive symptoms: **wheezing**, difficult expectoration, irritative spasmodic cough, paroxysmal dyspnea and recurrent hemoptysis.

carried out solely to complete the investigation of unexplained symptoms or signs of pulmonary changes observed roentgenologically or of evidence of tubercle bacilli in the sputum. The inclusion for brief study of instances in which the provisional diagnosis of tuberculous tracheobronchitis was not confirmed bronchoscopically permits a basic comparison with a group of patients proved to have this complication.

5. Kernan, J. D.: Tuberculosis of the Trachea and Main Bronchi, Ann. Otol., Rhin. & Laryng. **46**:763 (Sept.) 1937.

6. McIndoe, R. B.; Steele, J. D.; Samson, P. C.; Anderson, R. S., and Leslie, G. L.: Routine Bronchoscopy in Patients with Active Pulmonary Tuberculosis, Am. Rev. Tuberc. **39**:617 (May) 1939.

7. Hawkins, J. L. H. Jr.: Tuberculous Tracheobronchitis, Am. Rev. Tuberc. **39**:46 (Jan.) 1939.

8. Pierce, C. E., and Curtzwiler, F. C.: Tuberculous Tracheobronchitis: Roentgen Pathology, Am. J. Roentgenol. **43**:153 (Feb.) 1940.

9. Warren, W.; Hammond, A. E., and Tuttle, W. M.: Diagnosis and Treatment of Tuberculous Tracheobronchitis, Am. Rev. Tuberc. **37**:315 (March) 1938.

Tables 1, 2, 3 and 4 reveal the essential findings in the 58 cases in which tracheobronchitis was not found. The division of the material into groups A and B (table 1) at the time of the initial bronchoscopic examination was made the basis for therapy of the parenchymal lesion, collapse measures being used when a cavity was present or suspected. No case was considered abacillary until complete studies of the sputum, including repeated smears and cultures of concentrated specimens and animal inoculation with gastric washings, were made. The search for

TABLE 3.—*Status of Fifty-Eight Patients at Time of Bronchoscopic Examination; No Tracheobronchitis Found*

	Group A, 30 Sputum		Group B, 28 Sputum	
	Positive 20	Negative 10	Positive 26	Negative 2
Effective collapse	4	1	2	0
Ineffective collapse	0	0	5	0
No collapse	16	9	19	2
Symptoms				
Present	9	10	13	1
Absent	11	0	13	1

TABLE 4.—*Status of Fifty-Eight Patients at Close of Study; No Tracheobronchitis Found*

	Group A, 30 Sputum		Group B, 28 Sputum	
	Positive 11	Negative 19	Positive 12	Negative 16
Effective collapse	2	3	0	14
Ineffective collapse	0	0	4	0
No collapse	9	16	8	2
Symptoms				
Present	5	1	4	2
Absent	6	18	8	14

the source of tubercle bacilli in the sputum included, in addition to the use of other special roentgen and fluoroscopic technics, laminographic section of suspected pulmonary areas, particularly the collapsed lobes of the lungs.

Nevertheless, in 23 instances the suspicion of cavitation could not be removed before bronchoscopic examination, while in 5 cases the cavity was found. Together, these form the 28 cases of group B. The chief contribution of this group to the study is the obvious fact that collapse therapy was the most effective measure in obliterating areas of disease from which the production of tubercle bacilli continued as a hazard. Of the 16 patients in the group (table 4) whose sputum was finally noted

to be free from tubercle bacilli, 14 owe this result to collapse measures. On the other hand, most of the 12 patients whose sputum continued to show bacilli were persons for whom effective treatment continued to be unavailable, or, if available, was not applied.

The 30 cases which comprised group A are notable in that no cavity was demonstrable at the time of bronchoscopic examination and none could be found subsequently. Twenty of the patients had positive sputum and 10 had negative sputum (table 3). The status at the close of the report (table 4) reveals that the sputums of 19 became negative, the conversion in the majority being spontaneous. Failure of the sputum to become negative was noted in 11 cases and was attributed largely to underlying tuberculous bronchiectasis, for which collapse therapy was not favored. Whether or not tuberculous endobronchitis beyond the limits of bronchoscopic vision also accounted for the persistence of bacilli in the sputums in the remainder cannot be determined. Undoubtedly, such processes occur, not progressing beyond small order bronchi, and many heal primarily with regression of the parenchymal disease.

The important symptoms for which origin in a tracheobronchial lesion was suspected are shown in table 2, and failure to obtain bronchoscopic confirmation of this surmise implies that the initial evaluations of the symptoms were faulty. Further examination of facts and comparison with the group shown to have tracheobronchial lesions disclosed this important difference: symptoms which did not prove to evolve from tracheobronchitis were of more recent onset, occurred with less frequency and intensity and disappeared in a large number of patients after having been present for a relatively short period. Apparently, the statement "All that wheezes is not tracheobronchitis" may be reemphasized. Nevertheless, whether the presence of disease in the lesser order bronchi also accounted for the more benign character of the symptoms is still to be considered in some cases.

The importance of control of the parenchymal lesion, once tracheobronchitis has been ruled out, is indicated from the simple analysis presented in the foregoing paragraphs. The value of collapse therapy when cavitation has occurred or is suspected is clearly shown. While tracheobronchitis necessarily complicated and in a number of instances forced modification of the treatment of pulmonary tuberculosis, the general principles of management were retained for the group of patients whose cases are to be analyzed presently. This group consisted of 79 persons in whom tuberculous tracheobronchitis was found on bronchoscopic examination—apparently healed in 6 and active in 73. While the healed lesions were represented by visibly narrowed and scarred segments of the tracheobronchial tree, the active condition was characterized by pathologic processes of all the varieties and grades of severity classified by other investigators. Except for a few instances in

which an isolated tuberculoma, ulcer or superficial tubercle was noted, the vast majority of the tracheobronchial lesions consisted of granulomatous infiltration, ulceration and necrosis and varying degrees of edematous or cicatricial narrowing. Biopsies were not made frequently, but whenever a specimen was adequate for histologic study, the tuberculous nature of the process was disclosed unfailingly. Correlation of the degree of symptomatic disorder with the extent of the pathologic change was generally noted. In many cases the associated pulmonary changes bore no similar relationship. For example, a pinhead opening to an otherwise occluded main stem bronchus was sufficient to permit complete aeration of the corresponding lung. In others, a little manifest pulmonary infiltration was accompanied by an extensively ulcerated and granulomatously infiltrated tracheobronchial segment. Needless to state, converse conditions were true. The distribution of visible lesions is shown in table 5. In the main the tracheobronchial disease was found on the same side as the dominant pulmonary process. Two hundred

TABLE 5.—*Distribution of Tracheobronchial Lesions in Seventy-Nine Cases*

Tracheal	7
Bronchial	
Right bronchus	20
Left bronchus	20
Both bronchi	4
Tracheobronchial	
Trachea and right bronchus	8
Trachea and left bronchus	12
Trachea and both bronchi	8

and thirty-one bronchoscopic studies were made of this group, and some of the patients underwent as many as nine examinations. While it seemed that definite conclusions could not be drawn as to the effects of bronchoscopic treatment on the course of the local lesions, the promotion of healing was nevertheless attempted in nearly all the cases in which the tracheobronchial process was active. The measures employed were chiefly chemical cauterization with silver nitrate solutions of 5 to 25 per cent strength, the majority of the patients receiving a solution not stronger than 10 per cent. Where the patency of bronchi was compromised by masses of granulomatous infiltration or curtains of fibrous bands, these were nibbled away carefully with a biopsy forceps and the bases cauterized, so that the bronchial airway was restored or improved. Varying relief from annoying symptoms, such as audible wheezing, irritative cough and spasmodic dyspnea, was thus given many patients and lasted for weeks with some. An interval of at least six weeks was then allowed to elapse before bronchoscopic examination or treatment was repeated.

These patients with tuberculous tracheobronchitis were divided into two groups, A and B, as before (table 1). The findings at the time

of the initial bronchoscopic examination are shown in table 6. Group A consisted of 55 patients without roentgen evidence of pulmonary cavitation, and it is to be noted that 24 were receiving some form of collapse therapy considered on the basis of roentgen evidence to be effectual, yet the sputum of all but 2 of these contained tubercle bacilli. Thirty-one were receiving no collapse treatment and 27 of these also had tubercle bacilli in the sputum. On bronchoscopic examination 3 patients gave

TABLE 6.—*Correlation of Sputum with Status of Pulmonary Therapy and with Symptoms and Evidence of Tracheobronchitis at Time of Initial Bronchoscopy*

	Group A, 55 Sputum		Group B, 24 Sputum	
	Positive 49	Negative 6	Positive 23	Negative 1
Effective collapse	22	2	3	0
Ineffective collapse	0	0	10	0
No collapse	27	4	10	1
Tracheobronchitis				
Healed	3	0	3	0
Active	46	6	20	1
Symptoms				
Present	42	6	16	1
Absent	7	0	7	0

TABLE 7.—*Status of Seventy-Nine Patients with Tracheobronchitis at Close of Study*

	Group A, 55 Sputum		Group B, 24 Sputum	
	Positive 21	Negative 34	Positive 12	Negative 12
Effective collapse	10	16	1	10
Ineffective collapse	0	0	8	0
No collapse	11	18	3	2
Tracheobronchitis				
Healed	6	25	4	8
Improved	6	9	4	4
Unimproved	9	0	4	0

evidence of apparently healed tracheobronchial processes, but the remaining 52 presented active lesions and received topical treatment as outlined in an earlier paragraph. The subsequent course of events is noted in table 7, and the relation of sputum conversion to the therapy for the pulmonary lesions and to the treatment and course of the tracheobronchial disease is derived. In 34 of these patients the sputum became negative, and in 21 it remained positive. In 25 of those whose sputum became negative, the tracheobronchial lesions have healed, and in 9 unquestionable improvement has been noted, while the parenchymal

processes continued to exhibit a favorable course and required no significant alteration of existing therapy. The conversion of sputum in this group is therefore attributed to the healing of the tracheobronchitis. On the other hand, of the 21 patients whose sputum remained positive at the close of this report, in only 6 did the tracheobronchitis heal in the course of observation. The persistence of tubercle bacilli in the sputum in these instances in which the tracheobronchitis was visibly healed and the pulmonary lesions remained controlled may be explained by the probable presence of active lesions in the lesser order bronchi, which were inaccessible to bronchoscopic treatment. In 6 other patients of this group some improvement was noted, and until more complete healing of the tracheobronchitis occurs in these, continued failure of conversion of positive sputum to negative is expected. Finally, the lesions of 9 patients were unimproved or worse. These represent extrapulmonary foci, such as laryngeal or enterocolitic, which progress beyond control despite arrest of the pulmonary process. In these patients ultimately serious, if not fatal, extension of disease into the pulmonary parenchyma occurred.

Group B comprised 24 patients in whom, as stated previously, cavitation was known or strongly suspected. At the outset (table 6), the tracheobronchitis was found healed in 3 and active in the remaining 21. Tubercle bacilli were noted in the sputum of all but 1, and 13 were undergoing some form of pulmonary collapse therapy, which was considered on the basis of roentgen evidence to be effective in only 3. No active treatment was being given for the pulmonary lesions of the remaining 11. The subsequent course of the disease in these patients is noted in table 7. The increase of cases in which the sputum was not found to contain organisms, from 1 at the start of the study to 12 at the close, took place by virtue of successful collapse therapy in 10, representing the continuance of effectual treatment in some and the successful revision of previously ineffective procedures or the institution of new measures in others. Healing of the tracheobronchitis occurred in 8 cases and distinct improvement was noted in 4. On the whole, this group of patients has fared well, when the fate of those whose sputum remains positive and in whom collapse measures were continued ineffectually or revised unsuccessfully is considered. Bronchoscopic studies of these patients revealed that healing of the tuberculous tracheobronchitis occurred in only 4, and that while some improvement was noted in 4 others, the remainder were unimproved or worse when their status was last determined. Serious extensions of pulmonary disease have already taken place in several, while the prognoses of the others continue guarded. The failure of arrest of the underlying parenchymal disease and the corresponding lack of healing of the associated tracheobronchitis again

account for the persistence of tubercle bacilli in the sputum and the ultimate hazard to the patient. It is equally evident that disappearance of bacilli from the sputum indicates control of both the pulmonary and the tracheobronchial lesions and that the appropriate continuance, revision or addition of collapse therapy has been the chief effective measure.

COMMENT

The management of associated pulmonary and tracheobronchial tuberculosis is a subject of considerable discussion. As stated previously, controversy centers about the wisdom of collapse measures for the pulmonary process and the value or necessity of treatment for the tracheobronchitis.

Most therapeutic programs for pulmonary tuberculosis are based largely on the presence of cavitation, for which collapse therapy is applied. If the disease in the parenchyma is purely infiltrative, collapse measures are ordinarily deemed not necessary and the sputum may not contain organisms. If evidence of tubercle bacilli in the sputum appears in the presence of an effective collapse or a controlled infiltrative lesion, tuberculous tracheobronchitis may be suspected.

In many instances it has been shown that tuberculous tracheobronchitis is essentially benign and heals spontaneously, that it is improbable that any therapeutic measure will prevent crippling fibrostenosis of bronchi in some patients and that regardless of treatment the prognosis in general can be altered little. While no definite proof is available for the effectiveness of the local measures we have employed, it is our impression that healing, while it may have taken place spontaneously, has been hastened in a significant number of cases and that distressing symptoms have been strikingly alleviated. This is particularly so in cases in which the bronchial airway was improved so that drainage of exudate, often bacilli laden, from the distal bronchopulmonary bed was facilitated.

The effect of healing of active tracheobronchitis is probably best discerned when the underlying pulmonary disease is not accompanied with cavitation. This is precisely so in the cases of the 55 patients forming group A of the foregoing study. Here, arrest of lesions in the tracheobronchial tree with disappearance of bacilli from the sputum occurred in a significant proportion without alteration of the pulmonary therapy being followed. Of note is the large number of cases in which collapse measures were continued effectively in the presence of active tracheobronchitis and not abandoned, as advocated by some. For example, Myerson [3c] concluded:

> If the tubercle bacilli which are found in the sputum no longer come from the cavity in the lung but come from the surface of the granuloma, then pneumothorax is of no value. One can conclude that the pulmonary cavity is already

blocked off from the bronchial tree when the ulcerogranuloma is seen in the branch bronchus or main bronchus. As soon as a bronchial lesion is observed, pneumothorax becomes superfluous, as it collapses all the functioning tissues unnecessarily.

The adoption of the latter view would have virtually assured the reopening of many cavities, particularly those which had not been collapsed sufficiently long.

The importance of control of the underlying pulmonary process in the presence of tracheobronchitis is better observed when parenchymal cavitation is present or suspicion of it cannot be removed. Collapse therapy is commonly shown to be the most effective measure for cavitary disease, yet its application when tracheobronchitis coexists is considered hazardous by some. In our own experience, the great majority of sputum conversions in such cases was noted when collapse therapy was successfully added or when ineffective collapse measures were effectually revised. Significantly, in most of these cases healing of the tracheobronchitis followed. On the other hand, when revisions or additions of therapy failed or were not available, the sputum continued to show bacilli and the tracheobronchitis did not respond to treatment. The poor prognosis in such cases can therefore be attributed largely to the inability to control the disease in the parenchyma, whereupon, quite naturally, the complication of tracheobronchitis continues to influence the general course of the disease unfavorably.

SUMMARY AND CONCLUSIONS

A joint report is given by clinicians and bronchoscopists of their experiences with 137 selected cases of pulmonary tuberculosis in 79, or 58 per cent, of which the diagnosis of tuberculous tracheobronchitis was confirmed. When tuberculous tracheobronchitis was not visibly present, the control of disease remained ostensibly a clinical problem of applying appropriate measures for the pulmonary lesion. On the other hand, when tuberculous tracheobronchitis was found, the problem was more complex. The study of the entire problem yielded the following conclusions:

1. In cases in which tuberculous tracheobronchitis is not present and in which parenchymal cavitation is noted or suspected, collapse therapy is the most effective measure utilized to control the disease. The persistence of bacillary sputum when the pulmonary disease is controlled and no visible tracheobronchitis can be found is attributed to tuberculous bronchiectasis in some cases and may be accounted for by tuberculous involvement of the small order bronchi in others.

2. When tuberculous tracheobronchitis is visible, the management of the coexistent pulmonary lesion is as important if not more important

than the bronchoscopic treatment of the tracheobronchial process. Once control of the pulmonary lesion is obtained, conversion of sputum and healing of the tracheobronchitis follows in most instances. On the other hand, the contrary is noted when control of the pulmonary process by collapse therapy or otherwise is not possible.

3. Bronchoscopic treatment should aim to alleviate distressing symptoms and facilitate the drainage of exudate from the distal bronchopulmonary bed. While no definite proof is available of the effectiveness of topical measures, these appear to hasten healing, however spontaneous the latter may be otherwise.

INCREASED SENSITIVITY TO BONE-CONDUCTED SOUNDS

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LOS ANGELES

The importance of a correlation between the sensitivity for air-conducted and that for bone-conducted sounds to the differential diagnosis of an auditory disability was recognized in the sixteenth century. Capivaccio, by means of a simple method of test, separated cases of deafness into two types—those in which the bone sensitivity was acute, with the air sensitivity lowered, and those in which deafness to both air-conducted and bone-conducted sounds was present. As a result of his observations he deduced that bone vibrations passed into the cochlea directly through the investing wall, while air vibrations took the route through the middle ear. There were, of course, no tuning forks at that time.

In 1827 the English physicist Wheatstone observed that when the stem of a vibrating fork was applied to the vertex the sound was heard as if it were located in the head. However, when the auditory canal on one side was occluded with the finger or filled with water, the bone sounds were definitely heard in that ear. This phenomenon of lateralization of bone-conducted sounds was later described by Weber and is usually associated with his name.

Some fifty years later the German otologist Rinne published the results of his investigations of the correlation between air and bone acuity in cases of conduction and perception deafness. He found that in perception deafness, as in normal hearing, the air vibrations delivered by the prongs of a tuning fork held at the external ear are heard louder or longer than the bone vibrations produced by contact of the fork stem on the mastoid. This result is usually noted as AC > BC or as positive. In conduction deafness, however, this result tended to be reversed, and was noted as BC > AC, or as negative. The test, while valuable, is not entirely satisfactory, because too many variables enter into it and because it cannot be employed successfully even as high as 1024. I [1] demonstrated one of these variables in the test by pro-

Dr. F. W. Kranz, of the Sonotone Corporation, lent the equipment used in this study.

Assistance was given to the research on hearing through a grant-in-aid by the Carnegie Corporation.

1. Pohlman, A. G.: Tr. Am. Laryng., Rhin. & Otol. Soc. **43**:257, 1937.

ducing a 256 fork with which the Rinne test on a person with normal hearing gave a negative result. Then, too, the result with the stem vibrations cannot be compared quantitatively with that obtained on using the prongs, either in terms of loudness or in terms of the duration of the hearing; so that, while the statement that AC > BC is quite correct, the reverse, BC > AC, refers to the fork tests and not necessarily to quantitative methods. Cases in which the bone sensitivity is greater than air sensitivity are relatively uncommon.

The pioneer work of Wien established the possibility of an absolute determination of air acuity by a telephonic method. It was not, however, until some years later that a simple method of producing electrical oscillations by means of a radio tube was devised and the type of audiometer in use at present was made available for the testing of sensitivity to air sounds. In 1923 Pohlman and Kranz [2] described an electromagnetic bone receiver which imitated the stem vibrations of the fork. This device not only made for greater accuracy in observing bone sensitivity but also extended the range of frequencies tested to equal that developed by the air receiver.

Kranz and I repeated the experiments of Wheatstone under test conditions and with quantitative methods. We found [3] that when the external auditory canal was occluded in a person with normal hearing there was a marked increase in the sensitivity to bone-conducted sounds. This was limited to frequencies of the range below 2048. This same phenomenon was also observed under conditions of a partial occlusion of the external auditory canal but was less pronounced. The test on filling the canal with water [4] showed a result surprisingly similar to that obtained on complete occlusion, although under the first condition the vibrations of the drum membrane are damped and under the second the drum membrane is flanked by air on the two sides, with no change in the air pressure. In other words, with water in the ear the condition is one of conduction deafness, while under occlusion the lowered acuity for air vibrations is dependent on a blocking of the canal and not on a damping of the drum membrane. However, both conditions reduce the sensitivity to air vibrations for high frequencies more than for low frequencies.

Prolonged bone conduction has been described as present not only under conditions of occlusion and water filling, as noted by Wheatstone, but also when the drum membrane is loaded with secretions, as in otitis media, and when an otosclerotic process has resulted in fixation of the

2. Pohlman, A. G., and Kranz, F. W.: Proc. Soc. Exper. Biol. & Med. **21**:335, 1923.

3. Pohlman, A. G., and Kranz, F. W.: Ann. Otol., Rhin. & Laryng. **35**: 113, 1926.

4. Pohlman, A. G., and Kranz, F. W.: Ann. Otol., Rhin. & Laryng. **36**:1, 1926.

stapes. The explanation of the phenomenon was based on the accepted mechanics of cochlear reactions to sound. This doctrine of cochlear reactions stressed the importance of creating a differential pressure on the two windows which would permit displacement of the perilymph between the oval and round windows and result in transverse vibrations in the cochlear duct, specifically in the basilar membrane. This interpretation was adapted to the mechanics of hearing bone-conducted sounds by assuming that the contraction and the expansion of the otic capsule produced pressure changes in the labyrinth liquid. These pressures would tend to produce compensation reactions at the two windows but would not create the displacements held essential to hearing if both windows yielded equally. It was therefore assumed that the oval window was more resistant than the round window, and accordingly the greater compensations at the round window would produce the same effect as the differential pressures on the two windows. If, now, the oval window were more than normally fixed, its compensation motions would be reduced and those at the round window would be exaggerated. This in turn would produce the condition of enhanced bone sensitivity, which not only should obtain where the vibratory reactions in the drum membrane are reduced but which should be most pronounced where the stapes is definitely fixed by reason of bony ankylosis. This interpretation has been adopted by Herzog [5] and von Bekesy.[6]

Kranz and I [7] investigated a few cases of conduction deafness due to fixation of the ossicular chain, and the measurements demonstrated that the patients did not display the phenomenon of prolonged bone conduction nor did they experience improvement in bone acuity on occlusion of the external auditory canal. One of these patients was tested for bone acuity by the fork method in comparison with a person of normal hearing under conditions like those in the examining room of an otologist. The deafened person heard the stem vibrations longer than did the normal person. However, when the tests were repeated in a silence room, the results were reversed. This led to the interpretation that the difference was due to the masking effect of adventitious noises on the hearing of the normal observer and that enhanced bone acuity is not a real phenomenon under conditions of stapes fixation. This finding was reported at the International Congress of Otology in 1928 [8] and was vigorously protested. However,

5. Herzog: Ztschr. f. Hals-, Nasen- u. Ohrenh. **27**:386, 1930.

6. von Bekesy, G.: Ann. d. Physik **13**:111, 1932.

7. Pohlman, A. G., and Kranz, F. W.: Quantitative Tests on Bone and Air Acuity in Paracusis Willisi: Interrelation of Audiometric Tests, Arch. Otolaryng. **3**:136 (Feb.) 1926.

8. Pohlman, A. G.: Proc. Internat. Cong. Otol., Copenhagen, 1928.

since that date it has been confirmed, seemingly quite independently, by a number of observers. In any event, the fact that the phenomenon of enhanced bone sensitivity was wanting under conditions in which it should have been most pronounced introduced a reasonable doubt concerning the validity of the accepted explanation of cochlear reactions to both air-conducted and bone-conducted sounds.

The observations made by Kranz and me were later confirmed by me [9] in an investigation of the acuity of hearing for air-conducted and bone-conducted sounds in a series of cases in which the Rinne test had been negative and a series in which the test had been positive. I found that true prolonged bone conduction occurred only in those cases in which the sound conduction apparatus was functional and in which the external auditory canal was occluded or the drum membrane damped by secretion in the middle ear. I also observed that the phenomenon was frequency limited.

The English otologists have proposed what is known as the "A. B. C." or "absolute bone conduction" test. This test is made by comparing the bone sensitivity of the patient with that of the examiner under conditions in which the external auditory canals of both are occluded. While blocking of the external auditory canal tends to eliminate the masking effect of adventitious noises in both perception-deafened persons and those with normal hearing, occlusion also produces enhanced bone sensitivity, especially at 256 and 512 vibrations per second. Accordingly, the absolute bone conduction test takes into account only the masking effect of adventitious noises in the patient with a more or less functional sound apparatus and not true prolonged or enhanced bone conduction. The designation "absolute bone conduction" is obviously a misnomer.

Recently, von Bekesy [10] has proposed another explanation which takes into account not only the requirements of a functional sound conduction apparatus but also the frequency limitations in the phenomenon. Briefly, the interpretation is as follows:

When a bone receiver is applied to the forehead, low frequencies tend to vibrate the skull as a whole. The heavy mandible is not integral with the skull but is separated from it by the temporomandibular articulations. As a result, the skull tends to vibrate against the mandible, or, to state it another way, the mandible lags behind the skull vibrations on account of its inertia. This condition produces a counterthrust of the mandible on the external auditory canal, which develops air vibrations. These air vibrations are conducted more effectively through the functional drum membrane and ossicular chain when the external auditory canal is occluded.

9. Pohlman, A. G.: Ann. Otol., Rhin. & Laryng. **39**:927, 1930.
10. von Bekesy, G.: Akust. Ztschr. **4**:113, 1939.

This interpretation, as opposed to the older idea of fixation at the oval window, seems to explain the result. However, it does not account satisfactorily for a similar increase in bone conduction when the drum membrane is damped by the liquid applied to it, because here the efficiency of the sound conduction apparatus is much reduced.

Fortunately, the von Bekesy explanation is susceptible to direct experimental demonstration, because if the lack of integration of the mandible with the remainder of the skull is responsible for the phenomenon, then the integration of the mandible with the skull should suppress the effect. In other words, the prolonged bone conduction with the bite opened should be suppressed when the teeth are clenched. However, no significant differences in the increased bone sensitivity are detected under the two conditions.

Further, if the vibratory lag in the mandible is indirectly responsible for the increased bone sensitivity, then overcoming this lag should eliminate the effect and increasing the lag should exaggerate it. This was tested in the following manner: Two bone receivers were connected in series with an audiometric source and so arranged that they could be made to react in the same or in opposite phase. One receiver was placed in contact with the upper teeth and the other against the lower teeth with the bite open. With the receivers in phase the mandible would be forced to vibrate in phase with the skull, and if the von Bekesy explanation is correct the phenomenon of frequency-limited increased bone sensitivity due to occlusion should disappear. With the receivers in opposite phase, the forced mandible vibrations should be exactly opposite to those of the skull and the thrust of the mandible against the skull increased; and the increase in the bone sensitivity due to occlusion of the auditory canal should, according to the von Bekesy interpretation, be exaggerated. It is unnecessary to reproduce the audiograms made in this test, because there was no significant change in the sensitivity or in the frequency limitations under either of the two conditions. This evidence may not be construed as favorable to the von Bekesy interpretation.

Seemingly, otologists need not congratulate themselves on their knowledge of physiologic acoustics when the observations made by Wheatstone in 1827 have not as yet been satisfactorily explained.

Case Reports

ACUTE LARYNGOTRACHEOBRONCHITIS IN A NEWBORN INFANT

Report of a Case with Recovery Following Tracheotomy

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A number of cases of acute laryngotracheobronchitis have appeared in the literature in recent years. This is a report of a case of recovery from this disease occurring in a 5 week old infant who required tracheotomy. The prognosis in such a case is unfavorable. This patient is the youngest to recover from acute laryngotracheobronchitis of those reported on in the contemporary literature.

REPORT OF CASE

History.—P. G., a white female infant of 5 weeks, was admitted to the Parkview Hospital of Pueblo, Colo., on April 22, 1941, with the chief complaint of difficulty in breathing.

History.—The patient was a normal, full term infant, whose birth weight was 3,390 Gm. (7 pounds 1 ounce). She was breast fed and showed normal progress until the fourth week, when a nasal discharge and diarrhea developed. A nipple shield had been used in nursing because of the mother's cracked nipples. The infant was examined by Dr. J. D. Geissinger, who observed Bednar's aphthae of the soft palate and nasal obstruction. The ulcers were treated with a 10 per cent solution of silver nitrate, and calcium caseinate (casec) was prescribed to control the loose stools. After three days of treatment the diarrhea was controlled, but the ulcerative areas of the palate were unimproved. The pharynx became red and swollen, and papules suggesting early impetigo appeared on the face. A deep ulcer was noted on the left margin of the anus. There was no evidence of respiratory difficulty on April 21, but during the ensuing twenty-four hours respirations became noisy and labored. The infant was hospitalized because of increasing obstructive laryngeal dyspnea.

Physical Examination.—The patient was acutely ill but well developed and well nourished. She had a grayish blue pallor and showed all the signs of air hunger, with a respiratory rate of 60 per minute. There were a loud, crowing inspiratory stridor and an expiratory wheeze. Deep inspiratory retractions of the suprasternal notch, of the supraclavicular spaces, of the substernal region and of the sides of the thorax were noted. The baby appeared toxic and fatigued but extremely restless. Several ulcerative lesions, half-pea sized, were noted at the junction of the hard and the soft palate. The adjacent soft palate was injected and swollen, and the pharynx was reddened but without ulceration or exudate. The breath sounds were noisy and numerous coarse rales were present throughout the chest, but no area of impaired resonance was heard. There were no localized areas of dulness to percussion. The heart tones were clear but rapid. Examination otherwise revealed nothing abnormal except for a deep anal fissure.

From the Pueblo Clinic.

Clinical Course.—Immediate tracheotomy was advised, but parental consent was refused. A smear and culture of material from the throat revealed hemolytic streptococci. The infant was placed in an oxygen tent with elevated humidity and sulfathiazole (*2*-[paraaminobenzenesulfamido]-thiazole) in 0.1 Gm. (1.5 grains) doses was administered every four hours. No relief was obtained. The child became increasingly fatigued and cyanotic, and on April 23, twelve hours after admission, consent was finally given for tracheotomy.

Operation.—The preliminary introduction of a bronchoscope to insure an unobstructed airway was thought advisable. Laryngoscopic examination revealed moderate inflammation with considerable mucopus in the hypopharynx. The supraglottic tissues were thickened and edematous; the vocal cords appeared unchanged but lacked motility with respirations. The subglottic tissues were deep red and bulging. A 4 mm. bronchoscope was inserted through the small posterior glottic chink, and considerable mucopus was encountered in the trachea and aspirated. The tracheal wall was normal except for moderate injection.

A low tracheotomy was performed, using a 0.5 per cent solution of procaine hydrochloride for local anesthesia. A vertical cutaneous incision was used, the ribbon muscles being separated by blunt dissection. Incision was made in the third and fourth tracheal rings and a no. 1 Jackson tracheotomy tube introduced with some difficulty.

The retractions ceased, and the infant became quiet after the establishment of an unobstructed air passage. Good entry of air on both sides of the chest resulted. The cyanosis was improved but not completely relieved, and the infant was returned to the oxygen tent for the next twenty-four hours.

Postoperative Course.—The patient's condition remained critical after operation. Administration of sulfathiazole in 0.1 Gm. (1.5 grains) doses every four hours was continued for the next three days, a total of 2 Gm. (30 grains) being given. The mother's milk was given by dropper until the baby was able to resume breast feedings on the second postoperative day. A blood count made on April 25 showed 3,820,000 red cells, a hemoglobin content of 12.3 Gm. per hundred cubic centimeters (Sahli) and 21,900 white cells, with 53 per cent neutrophils, of which 46 showed segmented nuclei, 44 mature lymphocytes and 3 monocytes.

There was considerable mucopurulent exudate in the trachea for the next three days; this was removed by catheter suction through the tracheotomy tube. The tracheotomy tube was changed daily, but all unnecessary manipulations were avoided. By the third postoperative day the infant's condition was much improved and the palatal ulcerations were healed. On April 29 (the sixth postoperative day) the laryngeal obstruction had subsided so that the tracheotomy tube could be blocked for short periods. A roentgenogram of the chest then revealed normal pulmonary markings, with no displacement of the mediastinum. After three days of partial tubal block, the cannula was removed on May 1. For the ensuing two nights the child evidenced a slight crowing but respirations were not labored. The tracheotomy fistula healed with little scarring, and she was discharged from the hospital on May 18. A rectovaginal fistula had developed, which complicated her recovery.

The infant was seen on May 22 and June 5, 1941, and was in normal health except for the rectovaginal fistula. Her weight then was 5,190 Gm. (10 pounds 13 ounces).

CONCLUSIONS

A case of acute laryngotracheobronchitis occurring in a 5 week old infant is presented. A possible contributing etiologic factor was erosion of the soft palate, caused by a nipple shield, with the subsequent development of Bednar's aphthae and a descending streptococcic infection. Laryngotracheobronchitis, while common in infancy and childhood, is rare in the newborn. Recovery in this case followed bronchoscopy, tracheotomy, repeated tubal aspirations and the administration of sulfathiazole.

702 North Main Street.

CARCINOMA OF THE LARYNX IN A GIRL AGED 15

ARTHUR C. JONES, M.D., BOISE, IDAHO

Quoting from James Ewing,[1] first paragraph:

A review of the literature on juvenile cancer reveals conflicting views regarding the incidence, course, and general significance of this group of diseases, and these conflicts are clearly referable to the difference in the age periods included in the material considered by the authors. The writers who included under juvenile cancer all cases occurring in the first three decades failed to be impressed by any special characters of cancer in these periods and were inclined to dismiss the subject as of no special interest. On the other hand, the authors who considered especially the tumors occurring in the earlier age periods and particularly those occurring in infancy and the first decade were uniformly impressed with many peculiarities of cancer in childhood. There is a marked increase in the incidence of cancer in the third decade, and, when all these cases are included, the facts of interest regarding cancer in infancy and childhood are submerged and overlooked. It is now very well known that in the third decade cancer occurs rather frequently and does not differ materially from similar diseases in adult life. Real interest, however, attaches to the malignant neoplasms which occur in infancy and childhood up to 10 or 15 years, when special factors of heredity, nutrition, and growth may be expected to express themselves.

Quoting again, this time from Hayes E. Martin[2]:

This relative infrequency of cancer in children is not a true index of its significance, however, for, while most childhood diseases have a low mortality rate, cancer in the young is a highly malignant and fatal disease, as is proved by the mortality statistics already cited by Dr. Dargeon.[3] Cancer of the head and neck is less than $\frac{1}{100}$ times as frequent in children as in adults.

Undoubtedly many children die of undiagnosed cancer with symptoms and findings which in an adult would be readily recognized as characteristic of this disease, and I want to emphasize the necessity of making a most careful examination of children for possible malignant growth. It is true that the younger the person the more malignant is the neoplasm, but, on the other hand, these highly malignant tumors of childhood are more radiosensitive than are the tumors of adult life.

REPORT OF A CASE

I am reporting cancer of the larynx in a girl aged 15. I was called to the hospital in consultation in the afternoon. I found a well nourished girl who was

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Read at the meeting of the American Laryngological, Rhinological and Otological Society, Los Angeles, June 16, 1941.

1. Ewing, J.: A Survey of Cancer in Childhood, in Dargeon,[3] p. 13.

2. Martin, H. E.: Cancer of the Head and Neck in Children, J. Pediat. **15**:363-371 (Sept.) 1939.

3. Dargeon, H. W.: Cancer in Childhood and a Discussion of Certain Benign Tumors, St. Louis, C. V. Mosby Company, 1940.

having great difficulty in breathing. This difficulty was both inspiratory and expiratory, and tracheotomy was performed immediately. It was impossible to examine her larynx because of dyspnea.

Later I elicited the following history:

She had been going to school until about four months previously, when she became so hoarse that she was unable to carry on her class work. No effort was made to secure a laryngeal examination. The child was not acutely ill, but she dropped out of school because of her difficulty in reciting.

At this time there was very little difficulty in breathing. After about two months she became short of breath, had difficulty in going up and down stairs and was unable to run and play with the other children. According to the parents, there was a gradual increase in the difficulty of breathing, and the child remained quietly at home most of the time. Not until her breathing difficulty became very marked was additional advice sought.

A few days after the tracheotomy an effort was made to examine the larynx indirectly. It was difficult to see the left vocal cord, and the right cord appeared to be shoved past the midline. Because of my inability to make a satisfactory examination of the vocal cord, the tracheotomy wound was reopened and the larynx was split. Examination showed a curved slitlike opening between the two cords, about the size of the lead in a pencil. There was no cancerous-looking tissue on either cord. The mass that pushed the cord over was firm and extended up to the arytenoids and laterally about ½ inch (1.27 cm.).

Through the cooperation of the Union Pacific Railroad and Dr. Chevalier Jackson, the child was sent to Philadelphia for further examination.

Dr. Jackson's report is as follows:

"I regret to state that our records do not make it clear just where the specimen of tissue was taken for biopsy in the case of Miss Irons. This point is generally mentioned on reports, but this one simply says 'Larynx inspected with the smooth tipped anterior commissure laryngoscope and specimens removed for biopsy'."

The pathologist (Dr. F. W. Konzelmann) reported as follows: "Microscopic examination of the numerous fragments presented reveals on some a covering of stratified squamous epithelial cells. The layer is very much thicker than normal and many of the rete cells have a columnar outline. Directly beneath the epithelium there is a broad inflammatory zone rich in leukocytes and capillaries. Beneath this zone there is a tremendous mass of atypic epithelial cells. They are arranged in closely packed islands. The individual cells show marked variation in size and shape and staining reaction. Many bizarre mitotic figures are encountered, and many of the cells possess huge nucleoli. There is a small amount of dense stroma. A similar picture is encountered in all the fragments removed, except that some are not covered on the surface by adult epithelium. Diagnosis: squamous cell carcinoma (grade 4)."

The child returned, with the advice that roentgen treatment be given. The following report from Dr. A. M. Popma, the roentgen ray technician, gives the amount of roentgen radiation which was administered:

"From Jan. 25, 1938 to May 17, 1939 roentgen therapy was given to this patient at St. Luke's Hospital. A total of six courses of treatment was given, with six to eight weeks between courses. Multiple small ports, varying from 4 by 6 cm. to 6 by 10 cm., were used about the neck, an attempt being made to cross fire the larynx. The factors used were 200 kilovolts, 50 to 80 cm. target-skin distance, 8 milliamperes and filtration varying from ½ mm. copper 1 mm. aluminum to 2 mm. copper 1 mm. aluminum.

"As a rule one area was treated per day, and the daily dose varied between 100 and 250 roentgens. The total dose delivered to the skin over the combined fields and including the backscatter was 33,008 r. This was all given over a period of seventeen months. Considerable erythema of the skin developed, and for some time epitheliitis with vesicle formation was present.

"During the course of treatment there was progressive diminution of the size of the tumor. In spite of the large doses of roentgen rays to the cervical region, at no time did the patient exhibit any signs of hypothyroidism."

The roentgen rays did not cause a marked skin burn. Indirect examination showed a marked recession in the mass. The position of the cords became more nearly normal, and she was able to breathe for a few hours at a time without the tube. She could breathe with the tube closed, but she was always fearsome of being unable to breathe in the normal way and would not permit the tube to be left out for more than a short time. Because of the difficulty of reintroducing the tube, it was almost impossible to secure her permission to remove it. Consequently, she wore the tube until the time of her death.

In June 1939, two years and three months after her first admittance, she was again admitted to the hospital, with the following complaints:

She was unable to walk well. There was weakness of both legs, especially of the right leg. The onset of this weakness, about one month previously, was sudden, and the condition became progressively worse. There was no pain in the legs. She also complained of some difficulty in starting urination. She walked with an ataxic gait, with feet well apart, and had a tendency to fall to the right, and the entire body was tipped forward from the hips. The legs appeared to be spastic.

The pupils were equal and regular and reacted promptly to light. She had some difficulty in convergence. The fundus was normal. No other cranial nerves seemed to be involved.

The reaction of the muscles of the upper part of the trunk was negative. The epigastric and abdominal reflexes were negative. There was disturbance of sphincter control of the bladder and weakness of the quadriceps muscle in the right leg. The patellar reflexes were hypersensitive, and a patellar clonus was present. The Achilles tendons were bilaterally hypersensitive. There was ankle clonus. The Babinski sign was present bilaterally. Sense of position of the toes of both feet was absent. There was no disturbance of temperature or of sense of touch, pressure or pain and no muscular atrophy.

Examination of the blood resulted as follows: The white blood cells numbered 9,600 and the red blood cells 4,200,000; the hemoglobin content was 72 per cent; no pathologic cells were found. Roentgenograms of the chest showed nothing abnormal. Roentgenograms of the spine showed no evidence of metastasis. There was slight decalcification of the skeletal structure of the neck, which was likely due to the large amount of roentgen treatment.

A spinal puncture was done and iodized poppyseed oil 40 per cent was injected into the canal. Fluoroscopic examination with the patient in the Trendelenburg position showed that the iodized poppyseed oil had descended to the level of the space between the first and the second lumbar vertebra. There was apparently complete obstruction at this point.

Three days later reexamination following the injection of iodized poppyseed oil 40 per cent into the cisterna showed the oil to move freely up and down the length of the spinal cord when the position of the patient was changed, and there was no obstruction.

The previous impression of a block had to be changed in view of these observations.

Another roentgenogram of the spine showed evidence of cervical and upper dorsal changes in the spine. There was a moderate amount of decalcification. No bony erosion was seen. The interpedicular measurements were well within normal limits.

The Wassermann tests of the blood and the spinal fluid were negative, as were all of the other tests of the spinal fluid. There was a general increase in the loss of movement of the legs. Also, a few days later there was a loss of the reflexes of the muscles of the upper extremities. The grip in both hands became very weak. All of the muscles of the extremities became weaker. The patient had difficulty in speaking. Her respiratory rate did not increase; the pulse rate went up to 120. The temperature rose to 106 F., and she died on July 28, just a month after her admittance to the hospital.

The report of the postmortem examination is as follows: "This is the body of a fairly well nourished young woman about 64 inches (162.5 cm.) in height and weighing approximately 105 pounds (47.6 Kg.). The body is warm, and rigor mortis is not present. The body is not embalmed.

"The scalp is clean. There are no abrasions or abnormalities present. The eyes, ears, nose and mouth are normal. A tracheotomy tube is in place low in the neck. The appearance of the skin of the anterior surface of the neck is dark and rather scarred.

"The thorax and upper extremities show no gross abnormality. The breasts are well developed. There is some excoriation and appearance of beginning decubitus over the lower part of the back and the buttocks. There are definite decubitus ulcers over both heels and both ankles. The external genitalia and the rectum appear normal.

"The patient was placed on her face and an incision made along the spine. The spinous processes and laminae were exposed from the atlas to the third lumbar vertebra. Laminectomy from the first cervical to the second lumbar vertebra was done. On exposure of areolar tissue surrounding the spinal cord dura, a definite engorgement of vessels was noted. These had the appearance of vascular thrombosis, especially in the upper dorsal and lower cervical region. When the dura was opened, the cord seemed soft and felt almost mushy in the upper dorsal area. There was no gross evidence of tumor in the vertebral bodies, laminas, extradural space or spinal cord.

"The skull was opened in the usual manner. The cerebrum was removed above the tentorium. The tentorium was then incised and the pons cerebelli and medulla freed from attachments. The spinal cord was removed through the foramen magnum attached to the cerebellum, pons and medulla, for further gross and microscopic study. No gross abnormalities of the brain were noted except the presence of oily fluid in the cisterna magna. The incisions in the scalp and back were closed and the body turned on its back.

"The thorax and abdomen were opened in the usual manner. There was no free fluid in the abdomen or the thorax. The liver was of normal size; the edge had a normal acute angle and the cut surface showed no gross abnormalities. The spleen was of normal size and consistency. The stomach, the gallbladder and the small and the large intestine showed no abnormality. The appendix had been removed. The fallopian tubes, ovaries and uterus appeared normal. The bladder was empty.

"The heart was of normal size. A normal amount of pericardial fluid was present. The valves were normal. No abnormalities of large blood vessels were

noted. The lungs were free in the thoracic cavity. No areas of consolidation or tumor masses were noted. The peribronchial lymph glands appeared normal and were not hard on palpation. Thymus tissue was small in amount. The upper portion of the trachea and larynx, including the base of the tongue, were removed from the thoracic approach by blind dissection. The vocal cords appeared thick and scarred. There was no gross evidence of carcinoma."

The gross pathologic diagnosis was: "diffuse myelitis of the upper dorsal and lower cervical portion of the spinal cord; some softening and necrosis in the lenticular and caudate nuclei."

The spinal cord and brain and the larynx were sent to Dr. Chevalier Jackson. Unfortunately, the larynx was lost, and I have no pathologic report on that.

A pathologic report on the spinal cord (Dr. E. E. Aegerter) is as follows: "Multiple sections of this cord stained with hematoxylin and eosin and by Weil stain show the specimen to be from the cervical and upper dorsal regions. There is a remarkable degeneration of the ascending columns in the posterior and lateral regions, with accompanying degeneration of the cells of the posterior horns. These lesions are entirely characteristic of a compression lesion located in the lower dorsal or lumbar region. There is no evidence of inflammation. Diagnosis: Ascending degeneration, probably due to a compression lesion in lower part of the cord."

CONCLUSION AND COMMENT

Any child with hoarseness should receive a very careful laryngeal examination, because it is now demonstrated that such a child may have carcinoma of the larynx.

One of the most interesting parts of this report is that which describes the finding in the spinal cord of ascending degeneration. This was definitely not due to a compression lesion, because there was no compression lesion found at the careful postmortem examination. It might be possible for some radiologist to help explain the cause of this degeneration.

CONGENITAL AURAL FISTULA

MEYER S. FOX, M.D., MILWAUKEE

Congenital aural fistula is not infrequently encountered, but its occurrence in 6 members of one family is of sufficient interest and rarity to warrant reporting. The 6 cases herein described clearly demonstrate the familial and hereditary features of this condition.

A review of the literature reveals that this anomaly was first recognized and reported by von Heusinger,[1] in 1864. Selkirk,[2] in a more recent review, thoroughly discussed all phases of this interesting congenital condition. His article included a report of 172 cases which he had examined. The incidence in a controlled group of his cases was 0.9 per cent. The majority of writers, however, have reported a lower incidence—about 0.2 per cent. Congdon,[3] in a study of Oriental races, found an incidence as high as 4 to 6 per cent.

The origin of these congenital fistulas is explained on the basis of faulty development of the first branchial cleft or of the six primitive tubercles which go to form the pinna. Stammers [4] presented in his paper two embryologic diagrams to support the theory that the defect is caused by imperfect fusion of the auricular tubercles. These views are also mentioned by Keith,[5] Barrett [6] and Schwartz.[7] The familial and hereditary tendency is shown in the occurrence of the condition among several members of the same family. In Selkirk's [2] series there was no case in which more than 2 children of the same family were involved. Ruttin,[8] Stammers,[4] Schwartz [7] and others reported on the familial tendency, but in no case were 6 members of one family involved as reported in this paper.

From the Milwaukee Children's Hospital.

1. von Heusinger, K. F.: Hals-Kiemen-Fisteln von noch nicht beobachteter Form, Virchows Arch. f. path. Anat. **29**:358-380, 1864.

2. Selkirk, T. K.: Fistula Auris Congenita, Am. J. Dis. Child. **49**:431-447 (Feb.) 1935.

3. Congdon, E. D.; Rowhanavongse, S., and Varamisara, P.: Human Congenital Auricular and Juxta-Auricular Fossae, Sinuses and Scars, Am. J. Anat. **51**:439-459 (Nov.) 1932.

4. Stammers, F. A. R.: Pre-Auricular Fistulae, Brit. J. Surg. **14**:359-363 (Oct.) 1926.

5. Keith, A.: Three Demonstrations on Congenital Malformations of the Palate, Face, and Neck, Brit. M. J. **2**:438-441, 1909.

6. Barrett, J. H. P. B.: Congenital Fistulae, Dublin J. M. Sc. **131**:179-184, 1911.

7. Schwartz, A. B.: Congenital Aural Fistula, Am. J. Dis. Child. **38**:1339-1340 (Dec.) 1929.

8. Ruttin, E.: Zur Frage der Fistula auris congenita und der Aurikularanhange, Wien. med. Wchnschr. **77**:1019-1020 (July 30) 1927.

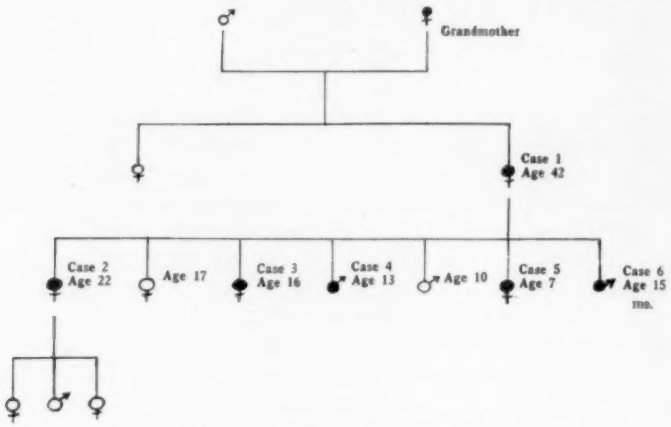

Fig. 1.—Family tree of patients.

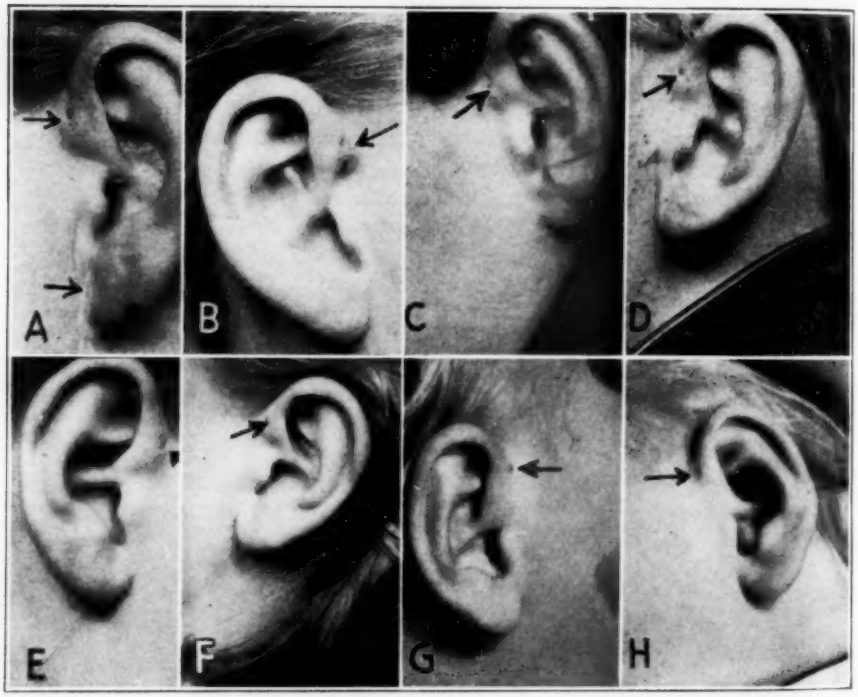

Fig. 2.—*A*, left ear, case 1; *B*, right ear, case 2; *C*, left ear, case 3; *D*, left ear, case 4; *E*, right ear, case 5; *F*, left ear, case 5; *G*, right ear, case 6; *H*, left ear, case 6.

The fistula presents itself as a small opening or dimple, usually located in the inferior portion of the ascending limb of the helix. Occasionally, it may be found in other parts of the helix, in the lobule of the ear or on the preauricular or the postauricular area. About 20 per cent of the patients affected showed involvement of both pinnas. In depth and extent the fistula varies from a mere dimple to a tract 20 mm. or more in length. The condition usually goes unnoticed, as symptoms are absent unless complications arise. However, when the opening becomes closed, a cyst forms and infection usually occurs. The sinus tract is lined by stratified squamous epithelium and contains sebaceous glands which may give rise to a milky discharge and incrustations. When blockage and infection arise, palliative measures may fail to bring relief, and unless the attending surgeon is familiar with the underlying congenital deformity, complete cure will not be effected.

Six cases of congenital aural fistula, those of a mother and 5 of her children, are reported here. The family tree of the patients is illustrated in figure 1. The individual photographs and histories relative to the fistulas are self explanatory.

REPORT OF CASES

CASE 1.—Mrs. J. W. (fig. 2 *A*), aged 42, the mother of 7 children, 5 of whom had congenital auricular fistulas, had a fistula located on the ascending limb of the left helix. During the patient's childhood a small cyst just below the lobule of the ear was removed. She stated that her mother had a small fistula of one ear.

CASE 2.—Mrs. E. L. (fig. 2 *B*), aged 22, the mother of 3 children, had a fistula of the right ear which required surgical correction during childhood because of cyst and abscess formation. No symptoms have arisen since surgical correction.

CASE 3.—V. W. (fig. 2 *C*), aged 16, had a fistula of the left ear. At the age of 5 a cyst formed and suppuration followed, requiring surgical correction. Since that time no symptoms have been present.

CASE 4.—R. W. (fig. 2 *D*), aged 13, had a small fistula of the left helix. At times there was a milky secretion from the opening, but otherwise he had no symptoms arising from the fistula.

CASE 5.—J. W. (fig. 2 *E* and *F*), aged 7, had bilateral fistula of the helix but no symptoms arising from these tracts. She has had numerous attacks of suppurative otitis media. The tract on the right side was 14 mm. in length.

CASE 6.—D. W. (fig. 2 *G* and *H*), aged 15 months, had bilateral involvement. He also had a port wine nevus of the right cheek. There were no symptoms from the fistulas.

SUMMARY

Six cases of congenital aural fistula, those of a mother and 5 of her children, are presented. The congenital development and hereditary features are described. In 3 (50 per cent) of the patients the fistulas closed and the complications which developed required surgical correction. A review of the literature indicates that this high familial incidence of congenital aural fistula is most unusual.

FOREIGN BODY (TOOTH) IN THE PHARYNX

ARTHUR H. DAVIS, M.D., TULSA, OKLA.

Cases of a foreign body in the pharynx are fairly common. Usually it is a particle of bone (fish bone) or a bristle from a toothbrush, which, embedded in the tonsil or the pharynx, is easily removed.

The following case is believed to be of interest.

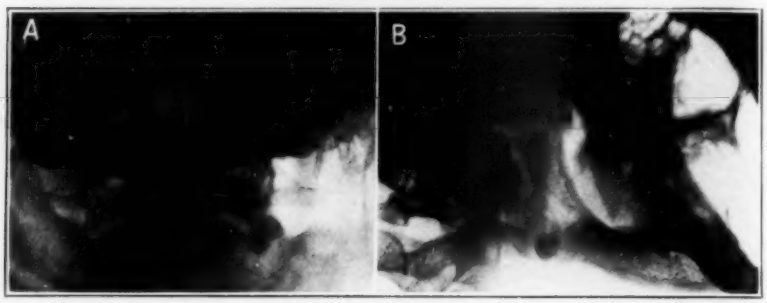

Fig. 1.—Roentgenograms showing a foreign body deep in the pharynx. *A*, anteroposterior view, and *B*, lateral view.

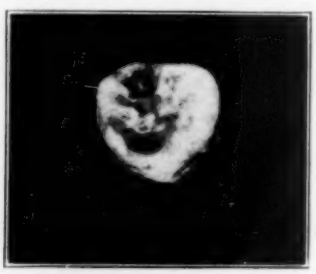

Fig. 2.—Photograph of the tooth after removal.

REPORT OF CASE

Mr. H., aged 52, was first seen May 26, 1941, having been referred to me by his oral surgeon. He complained of pain in the left side of his throat and difficulty in swallowing. This was the second attack since December 1940.

History.—In December 1940 a left lower molar tooth was extracted. Since that time he had had two infections of the throat simulating quinsy. His dentist had mentioned the possibility that part of the tooth was in his throat.

The following history was given by the oral surgeon who performed the extraction.

"With the throat of the patient well packed, the left third lower molar tooth was prepared for extraction. A chisel was placed against the tooth and lightly tapped. The upper half of the tooth promptly disappeared."

Careful searching failed to reveal the tooth. Roentgenograms revealed a foreign body deep in the pharynx.

Operation.—With the patient under general anesthesia, the left tonsil was removed by dissection. It was considerably swollen and adherent.

After removal of the tonsil a mass was felt in the tonsillar bed; further exploration with tonsil scissors revealed the object (tooth), which was removed.

Since the operation the patient has had no further trouble.

COMMENT

I am unable to explain how one could drive part of a tooth deep into peritonsillar tissue by tapping with a chisel, but this is apparently what happened.

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Clinical Notes; New Instruments and Technics

RECEPTACLE FOR READY-MADE COTTON WOOL EAR WICKS

Tse King, M.D., Peiping, China

Sterile ready-made cotton wool ear wicks are always useful in the practice of otology. To keep the wicks from forming clusters or being stuck together, they may be arranged on the corrugated paper which is a common packing material for fragile things, and so easily procurable. The procedure is simple: A swab is

Method of arranging ready-made cotton wool ear wicks in a row on corrugated paper.

made in the usual way, i. e., by twisting or rolling a thin metal applicator, held between the right thumb and index finger, into a small, thin sheet of cotton wool held firmly between the left thumb and index finger. Now, instead of being withdrawn from the applicator at once, the swab is transferred directly onto a groove of the corrugated paper, cut into convenient sizes. While the applicator is being withdrawn a little pressure from the left thumb or index finger applied to the cotton will make the wick stay in position. Several layers of the corrugated paper thus filled may be covered with cellophane or paper before being piled and wrapped together for autoclaving.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

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FUNCTIONAL EXAMINATION OF HEARING

ALFRED LEWY, M.D.
AND
NORMAN LESHIN, M.D.
CHICAGO

A paucity of reports on hearing tests in the literature is more in evidence this year than ever. Discussion of tuning fork tests is almost nil. There is an increasing interest in hearing aids, as indicated by the larger number of articles on this subject in the literature. The question of conservation of hearing has also received considerable attention. The presence of war has brought about an increased interest in military medicine. Abstracts pertaining to the effect of war on hearing and methods for the protection of hearing make up an important part of this review. The contributions of foreign writers to the entire subject of hearing tests, deaf-mutism and conservation of hearing are few and difficult to obtain.

HEARING TESTS

Kobrak [1] presents a series of new hearing tests and a plea for the retention of tuning forks. An experimental comparison is made between audiometric curves secured by using the Western Electric audiometer 2-B.E. and results obtained by a new method, using standardized Struycken tuning forks. All tests were made in a silent room. The Struycken tuning forks are equipped with a small apparatus enabling the forks to be struck always with approximately equal intensity. The vibration time of these tuning forks is almost constant. The forks are calibrated according to the periods of hearing sensation. The normal periods of hearing sensation are determined by the average figures obtained by testing the hearing of normal people with each of the six tuning forks. The method used is that of serial testing. Each subsequent test of the series with the same tuning fork starts at the level of loudness which was arrived at in the preceding test just when the tone disappeared. The test is based on a well known experience that

1. Kobrak, F. W.: New Tests and Clinical Experiments on Hearing, J. Laryng. & Otol. **55**:405 (Sept.) 1940.

a fading tuning fork, having ceased sounding, not vibrating, sounds again when brought to the patient's ear after a short interval. Although this factor has been regarded as an important symptom of fatigue or neurosis, it is an essential physiologic law of hearing, detectable accordingly in the course of the regular diminuendo in the vibrating tuning fork. For an example the author describes the test using a tuning fork which normally is heard for one hundred and ten seconds. For the first test, the tuning fork is held at the ear until it is no longer heard, say seventy seconds. The second test begins at the seventy second period and again the fork is held until it is no longer heard, say one hundred seconds. The third test begins at one hundred seconds, and the sound is heard for a period of six seconds. Finally, the test begins at one hundred and six seconds, and so on until the sound of the fork is no longer heard. Two series of figures are noted, the first is composed of the first and subsequent thresholds, as 70/100/106. The second is made up of the primary and additional periods, as 70/30/6/0. Figures like this appear a little too drawn out, suggestive of a slight impairment of hearing, but do not imply any definite organic alteration. Figures such as 90/20/0 or 80/30/0 would be nearer normal. Only in a limited number of cases is the final threshold reached in the first test.

Curves of the periods of hearing sensation are made, resulting in different forms, some falling steeply, as 80/30/0, and others drawn out, or fading slowly, as 70/30/6/0. Three sections of the curves have to be distinguished, the first section covering about the first two thirds or three quarters of the period of hearing sensation, the third section, about the last tenth of the period of hearing sensation and the second section situated between the first and third sections. A more or less general protraction, or flattening, in curves of periods of hearing sensation gives evidence of greater or lesser activity in the process. A fairly long reading for the final threshold points more to a lesion in the middle ear or to an established cochlear lesion, while a penultimate, or a final period, protraction points more to an active process in the sensory nerve. A routine computation of the total period of hearing sensation of the deaf ear is made and is taken as the basic figure. The three parts correspond to three degrees of loudness and result from the peculiarity in the decay of vibration. These are the initial fall, a second period and the penultimate periods ending in the final threshold. The decay of vibration is different in the first stage as compared with the later ones. The first stages of great loudness are heard chiefly by the deaf ear and are often heard only in advanced deafness. Repeated tests do not give identical figures, but the curves, when graphically reproduced, show approximately the same characteristics. Old established deafness is characterized by steep curves; deafness showing active advance, by protracted curves. The degree of the protraction seems to correspond

with the activity of the pathologic process. Tuning fork tests are the only hearing tests in which the stimulation is gradual and decreases at an equal rate. Increasing stimulation is also of some clinical importance.

The principle of varying stimulation leads to a new technic in reading the audiometer. To overcome the difficulty in deciding the precise threshold value, a maximum and a minimum figure for each reading may be noted and the mean figure taken as the threshold. The point at which the patient first hears the tone when the intensity is slowly increased is the crescendo, or C, reading. The point at which the patient first hears this tone when the intensity is slowly reduced is the diminuendo, or D, reading. The double set of readings plotted in an audiogram for all the frequencies gives a crescendo-diminuendo band, the C/D band. This band has certain characteristics. Old established clinical deafness gives a narrow C/D band, and actively advancing deafness often gives a wide band. Broad bands are more definite in air conduction than in bone conduction, in which they seldom occur. Broad C/D bands are more definite in the lower frequency scale and appear only occasionally in the higher part of the scale. These facts point to a possible middle ear factor in the production of a broad C/D band. Protracted curves of hearing sensation periods and broad C/D bands often occur together.

If the C/D band varies only 5 to 10 decibels, a mean value can be taken as the threshold. In broad bands of 20 or more decibels, the threshold is found by starting at the lowest intensity of the C/D band and increasing the intensity by 5 decibels for five seconds. After an interruption the intensity is increased again by 5 decibels for five seconds and then interrupted until the patient hears the tone, perhaps after a latent period of two to five seconds. This point is taken as the threshold. This latent period is described as the phase of preacoustic perception and is a physiologic condition of hearing.

Other physiologic conditions of hearing might be found by using staircase increase and decrease of the stimulus. Starting at 10 decibels above normal, at 1024 double vibrations, the patient turns the intensity dial until the loudness of the tone appears to be twice as much and then again until it is double the preceding loudness, until a point, such as 80 decibels, is arrived at. These steps are then made by decreasing the intensity in a similar manner from 80 decibels to 10 decibels. More steps are usually required to descend than to ascend. The difference between the number of steps required to ascend and the number required to descend varies with different people. The types of difference seem to be always the same, but the difference may be either small or very marked. These observations concern a zone of medium and intensive loudness lying more or less distant from threshold. For conditions prevailing at or near the threshold a stimulus of 10 decibels above normal

at 1024 double vibrations is sounded for five to ten seconds and then quickly reduced to 5 decibels. The sound disappears but reappears after a few seconds, with slightly increasing intensity. The first disappearance is like an inhibition of hearing, and the subsequent reappearance is like a release of the inhibition. The release seems to be followed by a form of sensitization. A common explanation for these phenomena might be a voluntary adaptive sensitization resulting from the actions of the intrinsic aural muscles. This same factor may play a part in the staircase tests, in which more steps are required to descend from a high to a low degree of loudness than in the reverse process. The damping inhibition is excessive when the loudness of the tone is being decreased. There may be some connection between these findings and the periods of additional hearing sensation observed with tuning forks.

Other interesting phenomena are observed when both the audiometer and the tuning fork are used in binaural combined stimulation. If a stimulus just perceptible is applied to one ear, say the right, at 1024 double vibrations and at 15 decibels, and a tuning fork of the same pitch but struck with more force is held at the left ear, the left ear will be stimulated by a tuning fork of greater loudness which is fading. The tone will first be lateralized to the left, then will approach the center; later it will be heard equally well in both ears; finally, it will be slightly lateralized to the right ear and then will be completely lateralized to the right ear. When the sound first becomes lateralized to the right, the tuning fork and the audiometer are presented separately to the right ear. The tuning fork, the tone of which was disappearing during the binaural stimulation, will sound louder than the tone of the audiometer. There is some incongruity between loudness and intensity of tone when the ears are separately stimulated after the commencing stage of lateralization to the right ear. What happens during the short interval between cessation of the binaural stimulus and the commencement of the monaural stimulus is the same thing that occurs in the series of periods of hearing sensation with the use of Struycken's tuning forks. This also is suggestive of a secondary factor of inhibition and releasing disinhibition taking place in the act of hearing. This is another observation associated with differences of intensity and loudness. Audiometric readings in terms of loudness as opposed to intensity would be helpful. With faint sound intensities it proved to be difficult to gage readings in these terms. Tuning forks, however, can be calibrated in degrees of loudness with greater reliability. Kobrak has found from his experience that six or more gross degrees of loudness can be distinguished in tuning forks of good quality and that interval values can be reasonably well distinguished. Based on an estimated loudness scale from 0 to 6 degrees, a loudness curve similar to the more usual

curves of hearing can be made. With constant use reliable accuracy is obtained and the test can be done in a short time. These loudness readings, although not scientifically exact, are more conclusive from a clinical standpoint than an audiogram. This test is based on figures which are psychologic and not mathematical estimations and gives a good basis for diagnosis. In cases of deafness having a scientific or particular clinical interest, however, more elaborate tests with the audiometer and tuning forks should be done. No one method should be adhered to in preference to the other. Each test has its own particular value. A combination of tests will bring out various diagnostic points.

Kobrak discusses the frequent finding of only slight bone conduction losses with the audiometer, usually in characteristic cochlear deafness in an early stage. He expresses the belief that this is due to a middle ear insufficiency, which is another physiologic factor. In cases such as these there is also a wide variation in the degree of protraction of the curve plotted from the additional periods of hearing sensation, as found with the tuning forks. He mentions the wide variations of bone conduction in normal people observed by investigators and stresses the fact that this functional variation is constantly present and must be reckoned with. He believes that if one not only considers this normal variation but also takes into account the additional periods of hearing sensation as estimated with the tuning fork, a more accurate picture of the pathologic change is obtained. He suggests that one add in decibels the additional periods of hearing sensation to the loss in bone conduction, to obtain the total loss for bone conduction. If bone conduction is 30 decibels and additional periods of hearing 40, then the total bone conduction loss would be 70 decibels. This corrected bone conduction determination would correct the air and bone conduction relationship, which frequently is questionable and confusing in making a diagnosis. An average decibel loss for bone conduction may be made by totaling the loss for all frequencies tested and dividing by the number of frequencies used in the test. Those frequencies not heard are computed at the maximum decibel figure. An average loss of 10 decibels is within normal limits, a loss up to 20 decibels is slightly suggestive of cochlear involvement and a loss above 20 decibels is an increasing indication of generalized involvement of the cochlea.

Kobrak mentions the characteristic dip at 1024 double vibrations in the bone conduction curves of many patients with hereditary deafness in an early stage and of those who have a history of otitis media without any other manifest sign of hearing loss. There is a fall in the curve between 512 and 1024 double vibrations and a rise again between 1024 and 2048 double vibrations. He believes that the early lesion at 1024 double vibrations "points to a weak spot in the cochlea." The reason

it is found in the bone conduction curves and is not so definite with air conduction tests is that it occurs in the region of natural frequency of the ear, and the middle ear, being closely associated with that natural period, is able to compensate to some extent deficiencies in the sense organ.

Kobrak presents only the general principles of these psychologic tests and expects to present the clinical side in a later paper. He concludes that both tuning forks and audiometers should be used in any case of scientific or practical significance. Combined tests for routine use consume too much time. For routine diagnosis a short test with reliable tuning forks is sufficient. This should be based on approximate estimation of loudness values. Instead of a single threshold, several values are often met, especially with tuning forks. For deciding on a suitable hearing aid a short audiometric test is sufficient. These observations in general may help solve some of the discrepancies mentioned in the literature regarding the lack of agreement of the Weber-Fechner law with the conditions of hearing.

Hulka [2] presents the results of bone conduction tests performed in 30 cases, on 38 inflamed and 22 noninflamed ears. Seven of the patients had acute purulent otitis media for the first time and 23 had an acute exacerbation of chronic purulent otitis media. Except in 4 cases all the tests were performed bilaterally, giving a total of 193 bone conduction tests performed on 60 ears. The tests were performed whenever possible during the acute stage of the disease and again after the inflammation had disappeared. The 2-B Western Electric audiometer was used, and the tests were made in a room having about 20 decibels of extraneous noise. The following is the composite picture of the bone conduction movements as observed during and after the acute stage in 29 of the 38 inflamed ears: There is an increase in hearing of low tones and a decrease in hearing of high tones by bone conduction during the acute stage. The depression in hearing for high tones by bone conduction may be greater than appears on the audiogram, because the loss may be masked either by interference from the noninflamed ear or by a linear increase of bone conduction. As the inflammation lessens, there is a gradual decrease of the low tone hearing with a variable change in the hearing of the high tones during the same period, which may be more depressed, remain unchanged or show recovery. Before all inflammatory reaction disappears, there may be a period when the hearing of low tones is depressed below the reference curve obtained by testing the completely healed ear. During the latter period hearing for the high tones shows improvement. The striking feature of the

2. Hulka, J. H.: Bone Conduction Changes in Acute Otitis Media, Arch. Otolaryng. **33**:333 (March) 1941.

typical bone changes is the see-saw motion represented by the bone conduction curves for the diseased ears. This is not a simple process, as the action of force and weight with a fulcrum in between, but a complicated one, suggesting the interplay of a number of factors. Several types of atypical shifts of bone conduction curves were observed. These observations cannot be explained either by the Bezold theory of a stretching of the stapedial ligament or by the Knudsen-Jones theory of masking by noise. Hulka offers the following explanation: The acute inflammation in the tympanum blocks the round window, which causes increased bone conduction throughout the whole range of frequencies. The increase in the high tones is more than counterbalanced by the penetration of toxins through the promontory into the basal turn of the cochlea. This produces a depression of the conduction of high tones, while the conduction of low tones remains increased. The atypical movements of the bone conduction curves may be due to the absence of the effect of the inflammation either on the round window or on the basal turn in some ears or in certain stages of the inflammation. By observing the bone conduction movements in acute otitis media one can evaluate the progress and extent of the aural lesions.

Singleton [3] suggests using a cotton plug moistened with 5 to 6 minims (0.37 cc.) of glycerin in the ear not being tested, to reduce and eliminate offending noises which are usually present in an office. He also believes that starting well above the auditory threshold and gradually diminishing the intensity 5 decibels at a time, using the tone interrupter, familiarizes the patient with the sound and prevents fatigue and carry-over. The patient also recognizes his auditory threshold more quickly and accurately with this procedure.

Fowler [4] discusses thoroughly the subject of tinnitus aurium and presents some of his own observations and interpretations. He divides tinnitus into two fundamental types, vibratory and nonvibratory. Vibratory tinnitus is mechanical in origin and may be objectively estimated by using the loudness balance technic, which compares it with an applied tone or noise of known loudness. It is balanced with the sound used for comparison. Tinnitus is usually uncontrollable except by masking. It should be compared or balanced with a tone applied to the opposite ear. In many instances a sound of only a few decibels, less than 10, above threshold may be required to equal intense tinnitus in loudness. The frequency tone of tinnitus may be determined objectively by comparison with similar applied frequencies. The

3. Singleton, J. D.: Hearing Tests in the Office, Arch. Otolaryng. **33**:1041 (June) 1941.

4. Fowler, E. P.: Tinnitus Aurium in the Light of Recent Research, Ann. Otol., Rhin. & Laryng. **50**:139 (March) 1941; Head Noises, Arch. Otolaryng. **32**:903 (Nov.) 1940.

measuring sound should be applied intermittently, and preferably to an opposite normal ear or an obstructive deafened ear. The frequency of the two sounds must not coincide too closely, because they may then appear as one; or if the measuring sound is louder, only the measuring sound will be heard. The measuring sound, therefore, cannot be satisfactorily compared with the tinnitus to measure the frequency of the tinnitus. The nearer the applied sound approaches the tinnitus in loudness and frequency, the more accurate will be the measurements. Start by applying discrete frequencies alternately well above and well below the frequency tone or band of the tinnitus and then gradually narrow the spread between the two exploring tones until they narrowly bracket the frequency band of the tinnitus. The masking of tonal tinnitus often varies greatly with the different frequencies used for it. In some instances it is easily accomplished at all frequencies. In other instances, less numerous, the masking is greatest as the masking tone approaches the frequency band of the tinnitus. Neither the loudness nor the frequency of the tinnitus alone determines the ease of masking it. The presence of tinnitus is always associated with some degree of deafness. Hearing varies with changes in the loudness and with fluctuation of the frequency band of tinnitus. The frequency and loudness of tinnitus, however, do not necessarily coincide with the deafness in general. Even though tinnitus may be limited to a narrow frequency area, when it ceases, the hearing often improves, not only in this area but over a wider spread in the audiogram curve. If the lesion causing the tinnitus progresses to complete mass degeneration of the neural elements, the tinnitus, unless continued from other locations, will cease entirely and the hearing loss at the former tinnitus frequencies will not be recovered.

Nothing definite is known about fatigue deafness resulting from tinnitus or the irritations causing tinnitus. Tinnitus is usually faint and therefore well below the intensity required to cause acoustic shock. The tinnitus fatigue factor is slow acting and varies with the etiology of the tinnitus. Neurologic fatigue and hypersensitiveness caused by tinnitus are important factors in some people because they tend to accentuate the tinnitus and the annoyance it occasions and therefore also the deafness. An increase in the loudness of tinnitus is often attended by an elevation in pitch, and vice versa. Vibratory tinnitus is characterized by its audibility to the examiner and by the demonstration of beats between the tinnitus and an applied pure tone. Nonvibratory tinnitus seldom, if ever, has the timbre of a pure tone. It is really an illusion of a tone or combination of tones. Tinnitus is an evidence of life, and when it is present the recruitment phenomenon is also present. Difficulty in masking tinnitus may be due to the fact that the origin of the tinnitus is medial or central to the end organ. If this explanation

is correct, then there is a test for differentiating the types of nonvibratory tinnitus—peripheral, central and medial. Bilateral tinnitus similar in tone but unequal in intensity will tend to be heard solely in the ear in which it is the louder. The bilateral character can be detected only by masking out the tinnitus on the side on which it is the louder. Environmental noise, as well as the contralateral tinnitus, may mask out the tinnitus on the side on which it is the weaker. If environmental noise masks tinnitus with ease, the lesion is probably in the cochlea. If loud noise does not mask faint tinnitus, Fowler suspects that the lesion is in a large part central to the end organ. Tinnitus may be associated with dips and troughs in the audiogram threshold curve of hearing. Fowler pointed this out years ago and interpreted tinnitus as an irritation of the hearing end organ element which is preliminary to subsequent degeneration. Therefore, tinnitus, if sustained, is a definite sign of increasing or potential deafness. Fowler discusses all the phases of tinnitus thoroughly. These articles bring together the scientific thoughts and observations on this subject and should be read by all.

Steinberg [5] and his associates report the results of the hearing tests made at the New York and San Francisco world's fairs in 1939, in one of the exhibits organized by the Bell System. Some 550,000 photographic records of the test for musical tones were obtained. Age, sex and color were also noted, to correlate with the tests. The tests were made in sound-insulated rooms, each visitor screened from the others. Instructions and the test were given from a record through a telephone receiver, which the visitor held to his ear. Tones of five different frequencies were given in the following order: 440, 880, 1760, 3520 and 7040 cycles. For each pitch, nine sets of tones, either 1, 2 or 3, were sounded, each fainter than the preceding one. When the tones became too faint to be heard, the listener was unable to write the number of times the tone was sounded correctly, and so a measure of his hearing acuity was obtained. The copy the visitor kept was so marked as to enable him to tell whether his hearing was good, slightly impaired or impaired. The tests revealed a certain relationship of age to hearing impairments. Both men and women showed increasing hearing impairment with increasing age. For the high frequency tones, especially 3520 cycles, the effect is more pronounced in men than in women, but for low tones the opposite is true, though to a smaller degree. At 1760 cycles there is little difference between the hearing of men and that of women. At the lower frequencies the hearing of members of the lowest age group (10 to 19 years) is slightly poorer than that of members of the next older group. This is probably due to the greater difficulty of the younger

5. Steinberg, J. C.; Montgomery, H. C., and Gardner, M. B.: Results of the World's Fair Hearing Tests, J. Acoustic. Soc. America **12**:291 (Oct.) 1940.

children in understanding the test and writing their responses on the test blank. A hearing loss of 25 decibels at frequencies up to 1760 cycles, which is a beginning handicap, was found in about 1.5 per cent of the young people, or 3 out of 200, and in almost ten times as many of the oldest age range, 50 to 59, or every seventh person. High tone deafness, although not so important for understanding speech, is indicative of a progressive condition which may later involve tones of lower frequency. Some 6 per cent of the persons in the 20 to 29 age group showed a loss of more than 20 decibels for the 7040 cycle, whereas almost 50 per cent in the oldest age group, 50 to 59, showed it. This rate would probably be higher in the population at random and is possibly due to the better economic status of the visitors at the fair. A comparison of the tests made at both fairs showed no appreciable differences at the three lower frequencies and very little difference in the women at the two higher frequencies. However, men tested in New York had some 3 decibels greater acuity than men at San Francisco. There was some evidence that people of high economic status had better hearing than those of low economic status. No trends of hearing acuity in relation to time of day could be discovered in any age group. No difference in hearing between the right and the left ear was found. The percentage of people having a hearing loss of 25 decibels or more for the various frequencies is as follows: for all ages, at 880, a minimum of 4 and a maximum of 11 per cent; at 1760, a minimum of 3 and a maximum of 12; at 3520, a minimum of 12 and a maximum of 18, and at 7040, a minimum of 18 and a maximum of 22 per cent. These tests are considered quite accurate so far as the reaction of the visitor and the frequent checking of the apparatus are concerned. It can be considered as a good estimate of a cross section of the slightly better economic group of the population. For those interested, a study should be made of the many charts and graphs analyzing each frequency, the average loss for each frequency in the respective age groups and the differences found in men and women. No conclusions or summary of the data is given other than that in the charts. Certain results of the survey are expressed in terms of aural canal pressure and equivalent free field intensity, and on this basis a comparison is made with results of other surveys of hearing.

Neville [6] presents the subject of senile deafness, with a review of the signs, symptoms and treatment of a group of 120 patients, whose average age was 66.9 years. The usual symptom was deafness, at times accompanied by vertigo and tinnitus. The vertigo of senile deafness was considered usually to be due to a lesion on the central pathways and in some patients to changes in the semicircular canals.

6. Thacker Neville, W. S.: Senile Deafness, M. Press **204**:282 (Oct. 9) 1940.

The hearing impairment usually started at the age of 50 years, with an impairment at 2048 cycles. People with this beginning impairment lose the qualitative difference between sounds of musical instruments. They are able to recognize vowels but have difficulty with the consonants, because the consonants are complex and contain high frequency notes. A typical early symptom of senile deafness is the difficulty in hearing when two or more people speak at once. Saxen pointed out two forms of pathologic change in deafness associated with old age: (1) senile atrophy of the spiral ganglion and (2) angiosclerotic degeneration of the internal ear. Correlating these changes and clinical findings, one notes that the first produces the typical clinical symptoms of presbycusis and the second produces deafness only when the sclerosis of the blood vessels has advanced so far as to produce degeneration of the organ of Corti and collapse of the cochlear duct. This also is responsible for the shortened bone conduction found in senile deafness.

Neville feels that the treatment of senile deafness is worth while. Beside treatment for the reduction of uric acid or cholesterol in the blood and proper treatment of the arteriosclerotic vertigo and tinnitus, vitamins and estrogens should be prescribed wherever indicated.

Johnson [7] reports 46 cases of submucous resection, with audiometric studies. In 65 per cent (30) of the cases in which there was nasal obstruction requiring a submucous resection, there was either no impairment of hearing or only high tonal dips. In those cases in which there was some impairment of hearing, the improvement, as shown by audiometric examination, was not sufficient to warrant a nasal operation for the purpose of improving the hearing. Nasal obstruction per se does not cause a characteristic impairment of hearing, nor should a submucous resection be recommended for improving deficiencies in hearing.

Ersner and Saltzman [8] report 3 cases of familial nerve deafness, in granddaughter, mother and grandmother. The essential characteristics of these cases is that in all, the right ear was affected first; 50 per cent of the females of three successive generations were affected, while all the male members of the family possessed normal hearing. These features follow mendelian laws. The cases are an illustration of progressive analogous nerve deafness in three successive generations with sex-limited inheritance.

Falkenberg [9] reports 7 cases of syringobulbia. Four of the patients had hearing disorders. The functional findings consisted of shortened

7. Johnson, M. R.: Nasal Obstruction and Impairment of Hearing, Arch. Otolaryng. **33**:536 (April) 1941.

8. Ersner, M. S., and Saltzman, M.: Progressive Analogous Nerve Deafness in Three Successive Generations with Sex Limited Inheritance, Laryngoscope **51**: 241 (March) 1941.

9. Falkenberg, K.: Hearing Disorders in Syringobulbia, Monatschr. f. Ohrenh. **75**:242 (May) 1941.

bone conduction and loss of hearing for high tones—in some cases loss for both high and low tones. The impairment was on the side of the bulbar lesion or at least was more marked on that side.

Selfridge [10] gives a résumé of signs and symptoms of the various vitamin deficiencies and the principal food sources of each vitamin. He points out that deafness is not caused by any one thing but is related to all the factors involved in growth, namely, endocrine principles, electrolytes, amino acids and vitamins. Every one should realize that in the treatment of chronic progressive deafness of either the nerve or the conduction type time is a great factor. Treatment may have to be followed for years before improvement, if any, occurs. The complete cure of chronic deafness is, perhaps, beyond the realm of probability, and one is fortunate if the progress of the disease can be checked. The time to begin the prevention of deafness is six months before conception. Each parent should be taught that an optimum diet containing all the essential food factors is essential for a normal healthy child, and the pregnant mother should carry out the instructions as to such a diet. This same careful diet should be given to the child from infancy to adolescence.

Rosenthal [11] reports the results of the use of prostigmine in 30 cases of impaired hearing. In 24 cases the impairment was chronic and in 1 acute. In 5 the impairment had been present less than six months and in 2 of these it was very recent. In 23 there was associated tinnitus. Tests were made in an ordinary room, with a 6-A Western Electric audiometer, which was calibrated before each test. Efforts were made to avoid fatigue, and readings were rechecked several times. The treatment consisted of the application of nasal tampons saturated with a 10 per cent solution of mild protein silver for thirty minutes, followed by a nasal douching and suction, and an intramuscular injection of 1 cc. of 1 : 2,000 solution of prostigmine methylsulfate three times weekly. The number of treatments varied from a few to, in 1 case, 71, with the majority of the patients receiving from 24 to 36 treatments. No untoward symptoms were experienced from the use of the drug. Nineteen patients, 6 with acute and 13 with chronic impairment, showed objective improvement of hearing; 16 showed subjective improvement and 9 showed no improvement. Eleven showed objective improvement of hearing in all frequencies; 3 showed a loss in all frequencies and 16 showed a loss in some frequencies and a gain in others. Although 19 of the patients with chronic impairment showed some improvement on the chart, only 13 reported subjective improvement.

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10. Selfridge, G.: Present Status of Vitamins in Relation to Eighth Nerve and Conduction Deafness, Arch. Otolaryng. **34**:125 (July) 1941.

11. Rosenthal, M.: Use of Prostigmine for Impaired Hearing, Arch. Otolaryng. **34**:540 (Sept.) 1941.

The 5 patients with recent tinnitus and impairment, 1 with an acute condition, were relieved of the tinnitus completely. Eleven of the 16 with chronic tinnitus reported improvement. One patient with Ménierè's disease reported complete recovery. Rosenthal states the belief that prostigmine is of definite value for impairment of hearing of short duration and for some forms of tinnitus aurium. Patients with chronic deafness show a smaller degree of improvement. However, his patients do not show the high degree of improvement noted in the series of Davis and Rommel.

The article is accompanied by an audiometric graph for each case. No bone conduction determinations were made and no diagnosis of the condition given other than that of acute or chronic deafness. No further analysis of the graphs is given. In looking over these graphs one notes that the improvement in most cases is very slight; in several of the cases the deafness was associated with nasal disease; 2 patients had discharging ears, 1, perforation of the drum membrane and 1, congenital syphilis. The ages of the patients were from 2 years to over 60. No attempt is made to analyze the relation of the intensive nasal therapy to the possible slight improvement. We feel that the author's conclusions are unproved.

AUDIOMETERS

The panel discussion [12] on audiometers held at the meeting of the American Otological Society at Rye, N. Y., in May 1940 brought out some interesting information. Those entering the discussion on audiometers were Mr. Howard A. Carter and Drs. Arthur W. Proetz, Edmund P. Fowler, C. C. Bunch, Max A. Goldstein, Austin A. Hayden, Frederick T. Hill, Horace Newhart, Harvey Fletcher, Gordon Berry, Werner Mueller and William P. Wherry.

The audiometer with half octave determinations seems to be the best for clinical use. It will detect progressive deafness better than the one in which the intervals are too long. For scientific investigation an instrument with sweep range is the best, but for clinical purposes it may be inaccurate in the hands of people not highly trained and is also harder to calibrate. Masking is necessary in all bone conduction tests and in cases in which the air conduction difference in the two ears is 40 decibels or more. Some use a buzzer with an intensity of 80 decibels, which can be stepped down. Others use a Bárány buzzer, and one otologist uses a double stethoscope connected to a pressure tank sufficient to mask a tuning fork or bone conduction receiver when held on the vertex. There was a difference of opinion as to how complete bone conduction tests

12. Hill, F. T., and others: Information Please: Regarding Hearing Aids and Audiometers, Arch. Otolaryng. **33**:500 (March) 1941.

should be made. Proetz said that he takes bone conduction only in the middle range. Bunch claimed that a test at 512 double vibrations is sufficient and is done principally as a check for the Rinne and Weber tests, while others, for example, Fowler and Berry, test the entire range. There was also some disagreement as to the value of including the 64 double vibration tone in the audiometric scale for testing. Fowler stated the belief that this is unreliable in that the patient cannot distinguish between hearing and feeling and that all the information needed could be obtained at 128 and 256 double vibrations. Carter mentioned that to include 64 double vibrations would increase the cost of the audiometer. Bunch and Goldstein expressed the belief that it should be included. The necessity for checking of the audiometer at least once a year and for the use of a sound-proof room or booth for testing was brought out. The necessity for including higher tones than are now available on the audiometer will have to be determined by further clinical practice. The work of Crowe brings out the necessity for such an addition. The value of the monochord for testing high tones was brought out. The disadvantages of the tuning fork are that no definite intensity can be maintained unless the instrument is electrified and that there has been no standardization of tuning forks. The subject of the high cost of the audiometer was brought up, and the hope was expressed that with increased production the price will come down.

It is the opinion of Sjöberg [13] that the question of the manner of registration of air and bone conduction curves has become international. A review of the literature indicates a real chaos. It is desirable, if possible, to have an international congress draw up a concrete diagram for general acceptance. The majority of otologists probably use Fletcher's system (the author calls it "Flechter").

The next question is: "Shall we use a loud speaker or a telephone?" Most practitioners are using the telephone audiometer. An exception would be in cases in which an operation has been performed recently. For exact hearing tests one must consider, in addition to differentiation of pitch, the fatigue phenomenon and adaptation. Adventitious noise should also be measured, since it causes of itself a masking effect. L. Holmgren uses an electric fan in his sound-proof room as a constant source of adventitious noise. The question of carrying over of sound from one ear to the other must also be settled. Usually a Bárány noise apparatus is used. Voss uses a stream of water or air against the drum membrane. In the Sabbatsberg Clinic one ear is plugged with oropax. In illustrating the use of the loud speaker with oropax for masking, the author reports 4 cases with graphs, showing the similarity of the

13. Sjöberg, A. A.: Critical Study of Electro-Acoustic Test Methods and Choice of Amplifier to Improve Hearing, Acta oto-laryng. **28**:437, 1940.

results for the two ears, although one ear may be totally deaf or severely deaf for tuning forks for part of the entire range. It is evident that the audiometric test, at least with the use of oropax and the loud speaker, has distinct limitations. It requires sustained attention and an experienced operator, which rules it out for children and certain adults. Speech, tuning forks and audiometric measurements should be combined. They are not mutually exclusive.

Kinney [14] presents some of his deductions about audiometry after ten years of personal experience. He discusses the term "decibel" and shows how an improvement in hearing at the greater losses, as 50 and 60 decibels, had a different interpretation as far as actual improvement of hearing is concerned, from a similar improvement at 30 or 20 decibels. A 10 decibel improvement at 60 decibels to 50 means that the patient can hear the tone 900,000 times less intensely, whereas a 10 decibel improvement from 30 to 20 means that he hears that tone only 900 times as intensely. In both instances the hearing is improved 10 decibels.

The use of the group, or phonograph, audiometer for group testing and screening of subjects with normal hearing should be properly supervised. About 20 per cent of the children reported as having some hearing deficiency are later found to be normal. This is due to low intelligence, inattention, foreign bodies in the ear and canal and other causes. The reports on group testing appearing in the literature have stimulated a company manufacturing hearing aids to advertise that there are seventeen million deaf persons in the United States. This statement may be regarded as a result of the abuse of audiometers.

The pure tone audiometer tests one ear at a time. The test is an individual one and has three variable factors: the patient, the examiner and the audiometer. The audiometer, as acceptable according to the standards of the Council on Physical Therapy of the American Medical Association, should provide the seven octave tones on the C scale from 128 to 8192, with a maximum error of only 5 per cent at any one tone. Several makes have a half octave sweep for notes above 2048. The direct oscillator principle employed in building most audiometers can reduce the error factor to less than 1 per cent, and the life of the apparatus is increased. Several factors produce variable results as far as the patient is concerned. Kinney believes that there is a daily variation in the acuity of hearing of a given person; although slight, it is definite, and hearing is most acute in midmorning. There is also variation in air conduction hearing with changes in atmospheric pressure, especially in abnormal conditions of the eustachian tube. Menstruation affects hearing in females with normal hearing. This is true especially in women with

14. Kinney, C. E.: Uses and Abuses of Audiometers, Arch. Phys. Therapy **22**:94 (Feb.) 1941.

otosclerotic types of impairment. Although all these points have not been scientifically investigated, he believes that these changes do occur.

The third variable factor in audiometric testing is the examiner. Kinney has found as much as a 10 decibel variation in the results obtained by different examiners of the same patient. The factor of so-called after-hearing image must be considered. Most persons hear a tone or the spoken voice at a decibel loss level lower when the intensity is being reduced than when the intensity is started below the threshold and is increased. Kinney records a point midway between the so-called ascending and descending hearing levels.

Kinney suggests the need for more investigative work in binaural and spoken voice testing. It seems that a person can hear the spoken voice better binaurally than in his better ear when the ears are tested separately. This test can be made either by putting the patient in a low sound room with a loud speaker several feet back of him or by using two similarly calibrated ear pieces connected with the same audiometer. When a microphone connected with the audiometer is used for accurate measurement the whispered voice test shows that with a difference of 20 decibels or more in the average pure tone loss between two ears the whispered voice loss is less than the average pure tone loss in that ear. With the average pure tone loss the same in both ears, the whispered voice loss is essentially the same. No mention is made by Kinney as to how the average pure tone loss is figured in comparing it with the whispered voice loss. Although the methods for testing bone conduction are being improved, they are far from perfect. An adequate bone conduction receiver has not yet been devised. No one as yet has been able to test the inner ear without some part of the middle ear being set into vibration at the same time. The hearing in the opposite ear has been fairly well eliminated by masking. The question of the satisfactory frequency of the masking tone is still unanswered. Some say it should be at the same frequency, whereas others state that a 60 cycle hum is adequate for all frequencies. In either case the intensity must be greater than that in the bone conduction receiver. Kinney does not believe that the bone conduction receiver held in place by a slight wire head band gives a true estimate of the bone conduction. He suggests that an assistant should hold the receiver in place. He also feels that a simple audiogram does not give sufficient information for the proper fitting of a hearing aid. A complete history, an otolaryngologic examination and tuning fork tests should also be used in prescribing an adequate hearing aid.

Bunch [15] made a comparison of the results secured by the Rinne test as usually conducted with a tuning fork and those secured by

15. Bunch, C. C.: The Rinne Test and the Audiometer, Ann. Otol., Rhin. & Laryng. **50**:47 (March) 1941.

audiometric tests. The tone 512 double vibrations was used with the audiometer, and the weighted c-2 tuning fork. In the majority of cases, if there is a difference of more than 45 decibels between the bone and air conduction thresholds as determined by the audiometer, the Rinne test will be positive. If this threshold difference is less than 45 decibels, the Rinne test will be negative. If the difference is 45 decibels with a variation of 5 decibels either way, the Rinne test will be indeterminate, or neutral. The use of the audiometer for the Rinne test can eliminate at least two factors of error: that due to the constant decrement of the tuning fork and that due to possible variations in pressure and in the selection of the point of contact against the mastoid process, because the tonal intensity is maintained for each setting of the intensity dial and is not influenced by changes while the pressure is varied at will or while the optimum point of contact is located.

Stern [16] contends that a certain type of audiogram appeared in large numbers, corresponding to certain clinically diagnosed cases. The graphs, like roentgenograms, should be used only as an aid to diagnosis. Graphs with two peaks, for instance, c-2 and c-4 or c-3 and c-5, appear to be diagnostic of trauma. The significance of the audiometric control of hearing speech is established. Patients with speech defects frequently have hearing defects that may be discovered only by audiometry. Persons who are considered normal on the basis of whispered tests may have defects discoverable only by audiometry.

In otosclerosis the audiometric defects may occur at variable locations in the tone scale. The author finds, in accordance with the literature, that loss at c-5 is often the first indication of degenerative inner ear deafness, principally hereditary with toxic or traumatic contributing factors. More often acoustic, rather than mechanical, trauma may be the cause. Early recognition is desirable in such cases.

Mundt [17] briefly mentions some of the advantages of audiometry. He believes that it is a decided improvement and of great value in otologic practice, especially as a quantitative examination of the hearing. It does not replace the tuning fork test as a diagnostic measure and should be used with the Weber, Schwabach and Rinne tests. The author brings out the point that normal hearing is not necessarily the same for all people and that in those cases in which slight defects are found with no subjective symptoms noticed either by the subject or his friends, training and other means should be used to improve the hearing function. The value of determinnig high tone loss, the use of audiometry in industrial practice and the value of prescribing hearing aids are also stated.

16. Stern, L.: Diagnostic Significance of Audiograms in Deafness with Special Reference to "Dips," Ztschr. f. Hals-, Nasen- u. Ohrenh. **47**:150, 1940.

17. Mundt, G. H.: New Developments in Audiometry of Interest to the Otologist, Arch. Phys. Therapy **22**:92 (Feb.) 1941.

HEARING AIDS

Carter [18] reports on the progress and work of the Council on Physical Therapy of the American Medical Association regarding the requirements for acceptance of hearing aids. The Council is assisted by a group of consultants who have special training and experience in this field. Several joint meetings of the consultants and the manufacturers of hearing aids were held, and a better mutual understanding was arrived at.

The requirements for acceptance of hearing aids were reported in our review of last year.[19] The important points are essentially as follows: A unit from stock is submitted with all the information as to the model, type, voltage and current consumption and a graph of the amount of amplification in decibels at semioctave intervals of C from 256 to 4096. Tests for any peaks or valleys between these points, if peaks and valleys have been found, should be specified. A copy of the guarantee and written instructions and evidence of adequate facilities for servicing the instrument should also be available. Any one wishing a copy of these requirements may obtain it by writing to the Council on Physical Therapy of the American Medical Association, in care of *The Journal of the American Medical Association*. The requirements went into effect in January 1941. The data and the instruments are submitted to an independent unbiased investigator for proper testing and inspection. He will recommend either its acceptance or its rejection. This report is next referred to the consultants in a confidential communication, for their approval. It is then referred to the Council, where it is given consideration in accordance with regular procedure, and an abstract of these findings is then prepared and recommended for publication. If the decision is one of rejection, a copy of the report is first submitted to the manufacturer, who is given thirty to sixty days to make changes in the instrument if necessary, or to withdraw the instrument from the market. If the changes or improvements are not made, the abstract of the findings is then published. When the instrument is accepted, a copy of the report is sent the manufacturers, and when necessary, a week or two is given him to correct or eliminate any misstatement of facts. At the time the report by Carter was published, February 1941, twelve hearing aids had been accepted by the Council, seven of the carbon type, three of the vacuum tube type and two of the semiportable type. Six additional instruments of the vacuum tube type were still undergoing investigation. The consultants believe that a hearing aid should be fitted by an otologist after a proper examination, including an audiometric test, a tuning fork

18. Carter, H. A.: Present Requirements for Acceptance of Hearing Aids as Determined by the Council on Physical Therapy, Arch. Phys. Therapy **22**: 98 (Feb.) 1941.

19. Lewy, A., and Leshin, N.: Functional Examination of Hearing, Arch. Otolaryng. **33**:92 (Jan.) 1941.

test and a whisper and voice test. A physician is better qualified to analyze the needs of a deafened patient and also to test the efficiency of hearing aids when this is indicated than is a technician or a salesman. The Council edits a booklet entitled "Apparatus Accepted," in which audiometers, hearing aids and other apparatus which have been investigated are reported on.

In the discussion on hearing aids at the Nineteenth Annual Session of the American Congress of Physical Therapy, held in Cleveland, in September 1940,[20] Dr. Kenneth Day, of Pittsburgh, gave his impression as to the types of hearing aid indicated for different types and degrees of deafness. As a general rule, few people will tolerate a hearing aid until they have an average hearing loss of from 30 to 35 decibels, unless accuracy of hearing is essential from an occupational standpoint. Patients with a slight hearing loss, of from 24 to 35 decibels, prefer a vacuum tube aid, because it gives greater fidelity of sound with slight or moderate amplification. They do not tolerate a carbon aid well, because of the peaking and nonlinear distortion. An average loss of 35 to 60 decibels can be served very well with the carbon aid. This type is more durable and economical than the vacuum tube aid, and patients get used to the distortion and peaking. For hearing losses over 60 decibels, especially when the loss is not uniform, a high fidelity vacuum tube aid is preferable. In these cases the carbon variety will often amplify certain sounds to the point of pain and consequent cochlear damage. True selective amplification to match the hearing curve of the patient is not possible with either type of aid. However, overamplified frequencies can be cut down in the vacuum tube appliance. This is of great value in cases of high tone nerve deafness with good hearing for the lower frequencies.

Air conduction receivers are efficient in cases of perceptive or nerve types of impairment; including all those in which there is a positive Rinne reaction. Bone conduction receivers are more efficient for conductive types of impairment if there is good hearing for the higher frequencies. In combined types of deafness with a negative Rinne test, both air and bone conduction audiograms should be made. The type of receiver selected should be the one which best transmits the higher frequencies from 1024 upward. Patients with bilateral hearing loss of over 50 decibels (average) can increase their intelligibility, especially for conflicting sounds, by wearing a hearing aid wired for amplification to both ears.

Mr. L. A. Watson, in the discussion,[20] brought out the point that by emphasizing the harmonics of a frequency level that has been completely lost one can produce subjectively the illusion of hearing a tone

20. Day, K., in discussion on papers of Kinney,[14] Mundt[17] and Carter,[18] Arch. Phys. Therapy **22**:100 (Feb.) 1941.

which is actually not heard and give a remarkable increased intelligibility of sound. For example, a person with a complete loss of hearing at 1000 cycles will get the illusion of hearing this missing fundamental by emphasizing the second harmonics or 2000 cycles. Persons with a severe loss of hearing, with only a narrow perception range, can be made to hear the fainter and more moderate sounds without being harshly affected by loud sounds at close range if circuits in the vacuum tube aid are properly arranged to amplify only the more moderate sounds and not to be overaffected by very strong sounds.

The panel discussion [12] on hearing aids at the American Otological Society meeting was entered into by Drs. Proetz, C. Stewart Nash, Berry, John H. Foster, Fowler, Bunch, Harris P. Mosher, Hill, Kenneth M. Day and Fletcher and Mr. Carter. The important facts about hearing aids are as follows: First, a complete examination of the patient should be made by an otologist. The proper fitting of an aid can be done best by having the patient try several instruments and by testing each one with him. The otologist should not limit himself to one make. The salesman, however, does. The trial and error system requires patience and endurance. At times it is possible to fit a hearing aid from an audiogram alone. In obstructive deafness it is difficult. The choice of a bone or an air receiver must be determined by trial. Even though bone conduction may be prolonged, hearing from an air conduction receiver may still be better. Use of a hearing aid is indicated when the impairment is such as to handicap the patient. The wearing of a hearing aid does not improve hearing. It may be harmful if an intense amplifier is used over a long time, especially for the hearing of high tones, owing to acoustic shock. In binaural pure nerve deafness, the air conduction receiver is generally more useful on the better ear. If a bone conduction receiver is used, it should be changed from side to side, to avoid constant pressure on one place. However, in binaural deafness, if satisfactory results can be obtained from the poorer ear, it should be used, leaving the better ear free for unaided hearing. Sometimes a binaural ear piece is used, producing an alleged 20 per cent gain.

Sabine [21] discusses the duties and purpose of the Council on Physical Therapy of the American Medical Association and the appointed group of consultants on hearing aids. Their duty is to investigate as fully as possible hearing aids offered to the public. No attempt is made to rate the merits of an instrument on any absolute basis or to evaluate the relative merits of different instruments, since the hearing improvement in any case will depend as much on the type and degree of the impairment as on the characteristics of the instrument used. On the other

21. Sabine, P. E.: Acceptance Tests of Hearing Aids, J. A. M. A. **115**:1633 (Nov. 9) 1940.

hand, interpreting purely physical measurements in terms of hearing impairment is open to question. The investigation limits itself mainly to size, shape, weight, voltage, current drain of batteries and a consideration of the mechanical features and construction of the instrument. Electrical defects, such as excessive noise, "feed back" and "squeals" under normal conditions are noted. Measurements are made to determine the frequency range over which the instrument shows appreciable amplification of sound intensity. Articulation and intelligibility tests are made, using a standard syllable and sentence list spoken at conversational level at a distance of 5 feet (12.7 cm.) in a quiet room. These tests give a means of forming a qualitative judgment of the performance of the instrument under conditions of normal use. Advertising matter is also examined, to determine whether the claims for the instrument are exaggerated. Acceptance of an instrument does not mean that all hard of hearing subjects will be benefited by its use. It means that it has been examined by a competent and unbiased investigator, that it is made well, is free from serious and electrical defects and that it produces sufficient amplification of sound intensity in the frequency range required for the understanding of speech so that it may be rated as a practical aid to hearing. In order that a person may determine what type of instrument he should use and which one will best fit his hearing needs, he should consult a competent otologist. A proper diagnosis and analysis of his hearing impairment will enable the otologist to determine what type of aid should be used, whether air or bone, and also what portion of the frequency range requires the greatest amplification in order to supply best the patient's individual defects. Hearing aids today cannot be fitted with the same precision as spectacles. However, better results will be obtained through the friendly cooperation of the otologist and the hearing aid dealer than by the hit or miss method of trying everything in sight and buying on the basis of the most persuasive salesmanship.

Halsted and Grossman [22] discuss the question of the proper fitting of hearing aids. They discuss first the pros and cons of the fenestration operation versus the use of a hearing aid for the same disability. The advantage of the fenestration operation is that sound perception after operation is more natural and the patient does not have the bother of a hearing aid. The disadvantages are that the improvement may not be a lasting one, that the new artificial communication between the middle ear structures and intralabyrinthine and intracranial structures might be a source of intracranial complications from an incidental infection and, finally, that the operation alters the biologic condition of the

22. Halsted, T. H., and Grossman, F. M.: Some Problems Involved in the Fitting of Hearing Aids, New York State J. Med. **41**:352 (Feb. 15) 1941.

labyrinthine capsule by removal of tissue and by the plastic procedure. In cases in which the operation is unsuccessful and the fistula is closed by new bone, the patient's condition may be worse than it was before the operation. The disadvantages of the hearing aid are that sound is not as natural as with the fenestration operation and that the patient has to wear a conspicuous instrument. The advantages are that the patient will hear with the aid as long as the batteries and mechanism are good. No operative measures are necessary, and the psychologic reaction of the patient in the eventual failure of the operation does not have to be overcome. Halsted and Grossman, after weighing the pros and cons, are in favor of the hearing aid. They suggest that any patient who is to have an operation should first be fitted with a hearing aid and the actual gain in hearing established for comparison with the gain obtained through operation. This would give a more accurate evaluation of both procedures.

For the purpose of fitting hearing aids the writers classify hearing impairments under four heads. Class 1 is the typical middle ear conduction or middle ear impedance hearing impairment. The fitting of a person who presents this type of defect may be successful with a bone conduction device. If the air conduction receiver gives as good or better results, it is preferable. When there is a chronic suppurative process or a tendency toward recurrence of a discharge, the bone conduction apparatus should be used. This is the only type of hearing impairment for which a carbon microphone instrument can be used. However, the authors prefer the radio tube amplifier. Class 2 is the inner ear conduction or impedance hearing impairment. It is caused by pathologic alteration of the hydraulic system of the cochlea. An example of this is a rigid round window membrane which does not permit the basilar membrane to move normally, although sound reaches the inner ear without any pathologic impedance of structures peripheral to it. Fluctuations in hearing occur as described by Crowe in his cases of Ménière's symptom complex. Adams called this condition "glaucoma auris." Shambaugh described diplacusis associated with this condition. These findings are due to hydropic changes of the endolymph. The best hearing aid for a person with impairment of this class is a strong undistorted tube amplifier with a receiver fixed to an individual mold. Impairments of classes 3 and 4 are also known as perceptive deafness. In class 3 is abrupt high tone deafness with shortened bone conduction, positive Rinne reaction and lateralization of the Weber reaction to the better side. The audiogram shows abrupt perception loss for high frequencies. The compensation derived from a hearing aid depends entirely on the stage of the condition. A loss beginning around 2000 cycles may be compensated fairly well, whereas one beginning around 1000 cycles will be difficult to fit. The amplifier should accentuate the high frequencies.

The authors suggest an even stronger suppression of the low frequencies in order to prevent these from masking the remaining high tones. Class 4 impairment, of the perception type, is due to an insufficiency of the nerve tissue which conducts impulses from the organ of Corti to the higher centers. A patient with impairment of this type normally denies that he is hard of hearing, specifying his difficulty as a distortion of sound rather than a lack of sound. Discrimination of speech in large rooms with high reverberation is more difficult for this type of patient than for any other. The tests show perception deafness with the loss of sensitivity graduated to increasing frequency. There is a disagreeable loudness when a fork of 256 cycles is struck heavily and held to the ear. The reaction to tone above threshold is important. With increasing intensity some patients note a change of quality of tone; the tone becomes rough, and the loudness becomes disagreeable. A very little amplification by the hearing aid will produce from the patient the remark that the voice sounds natural but cannot be understood because it is not loud enough. When the loudness is increased, he may understand better, but then the quality is not good. The principles to be applied in fitting this type of patient are, first, that the nerve should be protected from getting too many impulses, and, second, that most of the impulses should be generated from the noise created by voice, background noises being excluded as much as possible. Hearing aids for these persons should provide protection from noise more than amplification of sound. A smooth instrument with a crystal receiver and fitted ear mold is recommended. The volume should be turned on as little as is required for the understanding of speech. Specific intensity amplification is also advisable for patients of this group.

The testing procedure employed by the writers includes the taking of a complete history and an otologic examination. A routine test with a tuning fork of 256 cycles is made. Audiometric tests for both air and bone conduction are made, not only for threshold but also at higher levels of intensity, to determine if there is a change in the quality of tone and if the patient can discriminate pitch. The patient is also examined for diplacusis. Intelligibility tests based on the principles described by Fletcher and Steinberg are made. These consist of the calling of numbers, sentences and syllables under standardized conditions. These various tests will place the patient in one of four classes, and the intelligibility tests will determine whether the use of an instrument should be recommended. The latter tests are repeated with the instrument on. Two rooms are used in testing. One room has a high reverberation and a medium background noise level, with a calling distance of about 20 feet (610 cm.), similar to the living conditions of the average patient. The other room is specially constructed for low reverberation. In this are the audiometer and a loud speaker connected

over an amplifier with a phonograph and microphone respectively. The calling tests are made with the human voice, which is more natural than the phonograph reproduction. After the patient has acquired a suitable hearing aid, he should also be encouraged to make use of lip reading. For those not able to be fitted, lip reading may be the only means of communication with the outside world. The fitting of a hearing aid can be done only by a person with special training. Acoumetrists should be trained and licensed as optometrists are.

Holmgren [23] is of the opinion that the hearing is not endangered if the hearing aid is without appreciable distortion and if amplification does not reach the pain threshold. On the other hand, he believes that the use of the hearing aid in severe deafness may stimulate the association centers and acoustic nerve tracts. Further, he is of the opinion that the formants of the consonants lie principally in the high frequencies, while the vowel sounds are mostly in the low frequencies. The intensity of speech is determined by lower tones.

The wearer of a hearing aid becomes accustomed to the sharp tones of the consonants, and then the amplification of these higher tones can be increased. Holmgren compared this with the wearing of eyeglasses of increasing strength.

The author believes that a person with severe deafness may become so dependent on his instrument that in order to hear better he will gradually increase the amplification to a point above pain threshold. Animal experiments have indicated that the basilar membrane may be damaged by loud sounds. It is a common experience for otologists to observe deafness in people whose work exposes them to noise. A few years' more experience with hearing aids may be necessary to answer this question.

Lempert [24] compares the result obtained from a fenestration operation with that obtained from the use of a hearing aid. He claims that the operation restores physiologic hearing function to deafened persons, whereas the hearing aid merely amplifies the spoken voice without improving the hearing function. The operation checks the further progress of the condition, while the hearing aid permits progress to continue unabated until the stage is reached at which neither the hearing aid nor surgical therapy is of any value. A successful fenestration operation will eliminate tinnitus. This cannot be accomplished with the hearing aid. The hearing resulting from the fenestration operation is very good for group conversation, gives a perfect reproduction of

23. Holmgren, L.: Can the Hearing Be Damaged by a Hearing Aid? Acta oto-laryng. **28**:440, 1940.

24. Lempert, J.: Endaural Fenestration of the Horizontal Canal for Otosclerosis, Laryngoscope **51**:330 (April) 1941.

the voice as it is normally spoken and is not dependent on the direction of sound. The hearing aid gives distorted tone, is confusing in group conversation and is dependent on the direction of sound. The operation changes the entire mental status of the patient and presents social and economic advantages which are not presented by the hearing aid or are deterred by the use of the aid.

The use of hearing aids cannot be compared with the use of visual aids. One is expected to hear conversation or sound directed toward him from any direction, even in sleep. Such hearing protects one in life's hazards and is necessary in daily economic life. Sight, on the other hand, is present only when one makes a special effort to look in the direction of the object one is trying to visualize.

Lempert holds that the fenestration operation is no longer in the experimental stage. In the hands of a surgeon with the proper training and technic the surgical risk is practically nil. Practical physiologic hearing can be permanently restored in about 80 per cent of properly chosen cases of otosclerosis. The surgical procedure should be judged by the successes and not by the occasional failures. Special training for this type of surgery is an absolute prerequisite for good results.

Schier [25] discusses the ear piece, its importance, requirements and manufacture. He believes that the increasing satisfaction in the use and comfort of hearing aid equipment is due in part to the increasing use of personal ear fittings. The ear piece not only delivers the maximum output of an instrument to the middle ear structures but also is of great importance in testing hearing and selecting hearing aids. A hearing aid selected on the basis of an audiometric reading or with a poorly fitted ear piece will give different results in most cases when tested with an ear piece which is made to meet the anatomic and conductive needs of the individual ear. The ear piece should be reasonably retentive to all movement of the head and body and should be small, light in weight and as inconspicuous as possible. The tip should be as long as can be comfortably tolerated; sound conduction must be assured by proper channel bore and relationship of tip terminus and orifice to the tympanum. The material should be thoroughly nonconductive, acoustically passive, compatible to tissue and unaffected by aural secretion.

The technic of taking an impression is described. The hairs of the ear are trimmed, a small plug of cotton having a silk thread attached to it is inserted deep into the canal and the meatus is lubricated with liquid petrolatum. The properly mixed plaster is run or teased into the canal, with the tragus slightly retracted, until the canal is filled, and

25. Schier, M. B.: The Earpiece—in Testing for and Fitting Hearing Aids, Laryngoscope **51**:52 (Jan.) 1941.

then the tragus is released. The plaster is then poured until it fills the remainder of the concha sufficiently to overlap the margins of the auricular cavities. After the plaster has set, the helix and anthelix are carefully peeled away from the impression. After this, the ear is cleansed. Schier has found by experimentation a material for ear pieces which tends to passiveness of sound conduction and which as nearly as possible fits the characteristics of the instrument. This substance, methyl metacrylic resin, is now enjoying wide popularity. He describes also how the ear piece is made. The insertion of the ear piece should proceed as follows: The tip first should be directed under the tragus and into the meatus. The upper portions are then slipped under the crura and crus, and the ear piece is finally pressed home with a slight retraction of the lower third of the ear above the lobe. The longer the tip of the ear piece the greater and truer the sound conduction. The tip should be made as long as the patient can comfortably tolerate. If it is too long, it can always be trimmed or shortened. It should be at least ½ inch (1.25 cm.) in length. Sound blocks and muffling should be checked. The tip may butt up against the wall of the canal or some secondary curvature. The mere enlargement of the orifice may be sufficient to overcome this. Mass is nonessential to successful retention of the ear piece.

Schier concludes that the diagnostic equipment used for prescribing aids in the future must provide for conductive mediums similar to those used with the instrument. The performance of the instrument will depend on the degree of perfection of the ear piece which conveys the possibilities and characteristics of the instrument to the ear. Good conveyance can be accomplished only through a molded ear piece made to meet the anatomic and conductive needs of the individual ear.

Carter [26] describes the physics of the vacuum tube aid in a detailed manner. His paper is illustrated by many drawings. He also defines various terms used in discussing sound and audiometry.[27] Frequency is defined as the number of cycles occurring per unit of time. A cycle is a complete set of the recurrent values of a periodic quantity. The bel is the change in the intensity level of a sound which results from a tenfold increase in the energy causing the sound. The decibel is one tenth of a bel. The phon is the unit of loudness level. At a frequency of 1000 cycles per second, the loudness level in phons of a pure tone is numerically equal to the intensity level in decibels as judged by listeners.

26. Carter, H. A.: Physics of a Vacuum Tube Hearing Aid, Tr. Am. Acad. Ophth. **9**:162 (Dec.) 1940.

27. Carter, H. A.: Decibels and Cycles, Tr. Am. Acad. Ophth. **45**:21 (May-June) 1941.

CONSERVATION OF HEARING

The committee on conservation of hearing of the American Academy of Ophthalmology and Otolaryngology reports a notable expansion of the public school health program in many communities, through provision for audiometric tests of school children.[28] Dr. Gardner, chairman of the committee on hard of hearing children of the American Society for the Hard of Hearing, reported for the year 1938-1939 that 1,877,031 pupils were tested in 767 towns and 126 counties, compared with 1,062,000 pupils in 233 towns and 22 counties reported two years previously. The Works Progress Administration has financed case-finding hearing surveys in several states when satisfactorily sponsored by local tax-supported agencies. This has disclosed large numbers of children in need of corrective treatment or educational readjustment. The greatest obstacle to more effective work is the lack of understanding among the majority of general practitioners and the laity of the elementary principles related to early detection, prevention and amelioration of hearing deficiencies. The committee urges the members of the Academy to use their personal influence in stressing this information through refresher and postgraduate courses as well as in the undergraduate curriculum. The committee also recommends the adoption of changes in terminology, such as the use of "hearing impairments" and "deficiencies" instead of deaf and deafness, as also recommended by the Committee on Audiometers and Hearing Aids of the Council on Physical Therapy of the American Medical Association.

The report of the committee on conservation of hearing of the American Academy of Ophthalmology and Otolaryngology [29] emphasizes the increasing importance of the conservation of hearing as a problem in the national health program. With the possibility that funds will be curtailed because of the national defense program, financial aid from other sources must be obtained to continue routine hearing tests for all school children. This procedure will effect many appreciable reductions in the number of hearing-handicapped persons in the population. The increased use of a simply constructed, quickly operated low cost pure tone audiometer with a fixed zero reference level especially constituted for screening purposes has led to the disclosure of early hearing impairments of the high tones, such as 4096 and 8192 cycles, not revealed by the ordinary phonograph audiometer. Tests of 10,000 school children in Minneapolis with the pure tone audiometer showed that deficiencies

28. Report of the Committee on Conservation of Hearing, Bull. Am. Acad. Ophth. & Otolaryng. **9**:175 (Dec.) 1940.

29. Report of the Committee on Conservation of Hearing of the American Academy of Ophthalmology and Otolaryngology, Tr. Am. Acad. Ophth. (1940) **45**:25 (July-Aug.) 1941.

for the higher tones were from two to three times as common among those of school age as defects for the lower frequencies alone. Hearing tests of larger groups made with the phonograph audiometer fail to disclose more than 50 per cent of pupils who have significant hearing losses as judged by the standards employed in clinical practice, using pure tone audiometers. This does not discredit the phonograph audiometer, which was developed as a means of rapidly screening large numbers at a minimum cost and was not designed as an instrument of precision. No criteria have been established as yet for determining the degrees of loss at different frequencies sufficient to justify referring the child for further otologic examination and treatment. New York, Pennsylvania, Oklahoma, Oregon, Indiana and Washington have laws pending for the discovery and reporting of deaf and hard of hearing persons, the conservation of hearing and the education and rehabilitation of those who are acoustically handicapped. Every legislative program should include the audiometric testing of all school children under the joint administration of the state board of health and the state medical association. Funds should be provided for a competent state supervisor, to insure uniformity in testing technic, recording and reporting of cases and proper follow-up. Competent medical care should be provided for indigent and medically indigent persons with hearing defects, on a basis which is fair to all concerned. Proper elementary instruction should be required in the public, teachers', hospital, nursing and general institutional schools. The committee recommends that hearing tests by acceptable audiometers be included in the preemployment medical examination for every applicant for a position and that they be repeated at suitable intervals.

There has been an improvement in the construction and appearance of electrical hearing aids, especially in those of the vacuum tube type. Hearing aids, multiple and single type, are being used by children in schools for the deaf and in the home. Seven carbon type and ten vacuum tube type hearing devices have been accepted or recommended for acceptance by the Committee on Hearing Aids and Audiometers of the Council on Physical Therapy of the American Medical Association. Acceptance has also been withdrawn from or refused to a number of manufacturers whose agents have violated the regulations for advertising their products by making exaggerated statements and unjustifiable claims. The Council also publishes lists of acceptable products in this line at various intervals in *The Journal of the American Medical Association*. The acceptance or rejection of audiometers is being delayed for further investigation. Several new simplified or improved models are reported to be announced soon. Notice of their acceptance, if this should materialize, will appear in *The Journal of the American Medical Association*. The Committee recommends that members of the American

Academy of Ophthalmology and Otolaryngology who assume the otologic care of patients become active members of the American Society for the Hard of Hearing and the American Association for Promoting the Teaching of Speech to the Deaf. Their publications contain articles of helpful information which can be passed on to patients. Every otologist, as a public-spirited citizen, should work toward the establishment of accurate testing of the hearing function as a regular feature of every periodic health examination. This will be an important safeguard to the hearing.

Newhart [30] presents facts pertaining to the conservation of hearing which he feels should have greater publicity, to accelerate activity in the solution of the problem. Some of the obstacles to progress in the field of prevention are the following: There is a widespread misconception that treatment for hearing impairments is futile. The incidence of significant hearing deficiencies among the population, as disclosed by the audiometer, is high. The use of the audiometer has shown how some of the older tests are inadequate to disclose early impairments and has shown the necessity for a quiet environment for testing and proper masking. There is need for education of educators and parents to appreciate the fact that a slight hearing impairment may cause such things as retardation, speech defects and behavior problems. The work of Crowe, Guild and other authors shows the necessity for early detection of hearing impairments and how improvement can be accomplished with proper treatment. The public is uninformed as to the possible causes of hearing defects due to disease elsewhere in the body. Finally, the word "deaf" is improperly used.

For effectively conserving the hearing of the population, the periodic testing of the hearing of all pupils with the audiometer should be incorporated in every school health program. The use of a recently available low cost pure tone audiometer applicable both for screening and for final tests has greatly reduced the cost of equipment for communities having a school population under 3,000. Conrad G. Selvig, chairman of the committee on legislation for the hard of hearing of the American Society for the Hard of Hearing, has outlined the following program for state legislation:

1. Compulsory hearing tests for all children at regular intervals by competent scientific methods.

2. Proper medical follow-up treatment and care for all in need, and instructions to parents as to its necessity.

3. Remedial instruction in the form of lip reading from primary grade through college. Special training in speech and voice where necessary. Group hearing aids in every school where there are five or more hard of hearing children.

30. Newhart, H.: Progress in the Conservation of Hearing, Ann. Otol., Rhin. & Laryng. **50**:129 (March) 1941.

4. Vocational guidance.

5. Adult classes in lip reading serving the hard of hearing in all communities in the state.

6. Assistance to the hard of hearing in search of employment and a fair deal under Civil Service rules.

7. A full-time director of work for the hard of hearing on the staff of the State Department of Education.

8. Training courses for teachers of the hard of hearing in every State Teacher's College.

Newhart suggests adding another requirement, namely, to safeguard and protect workers in industries which might cause damage to the hearing. Not all of these objectives can be accomplished at one time, but some start should be made, with at least the provision of regular audiometric tests of all school children to be administered by the department of education in cooperation with the state board of health.

A résumé of the Oregon program for the conservation of hearing is presented in the Transactions of the American of Academy of Ophthalmology and Otolaryngology.[31] This program has been evolved from the experiences of the other states as well as from that of Oregon. The local health officer explains the details of the program to the superintendent of schools, who arranges for suitable testing rooms and draws up a schedule for the test. Printed instructions are left with the superintendent, who in person explains the test to the teachers. Two types of test are made. One is a rough screening process, which may be repeated for certain pupils, and the other is the precise scientific measurement of the hearing of those screened by the preliminary test. The phonograph audiometer having forty receivers in series is used. As many as 120 children can be tested in a fifty-five minute period if proper preparations are made in advance. First grade pupils are tested in groups of 6 by the monitor method. The pupil whispers to the monitor what he or she hears in the receiver, and the monitor in turn writes it down. The children who fail to hear well are retested on the group audiometer the same day, and if they still show unsatisfactory results, are given a pure tone audiometric test at a later date. These children are also given a prompt medical study, to determine the cause, degree and prognosis of the defect. The school department observes the reactions, attitudes and achievements of the child. This is added to the medical report to permit a final decision on the educational needs of the child who is handicapped.

If the child's audiogram and medical inspection show a need for further otologic investigation, the parent is sent for and the situation explained. Parents are also given printed information regarding hearing

31. The Oregon Program for Conservation of Hearing, Tr. Am. Acad. Ophth. (1940) **45**:11 (May-June) 1941.

health and medical aspects of hearing. The public health nurse takes the hearing health history, obtains the name of the physician who is going to examine the child and gives a copy of the audiogram to the parent, to take to the physician. The financial status of the family is obtained, and if indicated some means for proper care is provided. If the parents do not come to school when asked, a nurse contacts them and gets the necessary information. The physician who later examines the child is asked to report on the diagnosis, treatment, prognosis and necessity for classroom adjustment if indicated. He is also urged to send in occasional reports on the child's progress. The school examiner also endeavors to recheck the hearing of the children with hearing impairment several months later. Patients who continue to show deficiencies and who do not receive medical attention are again checked by the nurse. These progress reports are important, because they show how many children went to physicians and how many of them improved and also how many did not receive medical attention and have remained the same, become better or grown worse. After succeeding calls and interviews with the parents the health nurse classifies the patients on the basis of follow-up, as follows: (*a*) children seen by physicians and receiving treatment; (*b*) children who have not as yet visited a physician but whose parents have promised to see that they do; (*c*) children whose parents gave no cooperation, and (*d*) children whose parents have been approved for medical relief by public welfare agencies.

After the report of the physician is obtained, the principal, the child's teacher and the public health nurse confer on the particular needs of the child. In some instances the teacher of special education, representatives from the state department of education or the superintendent of the state school for the deaf are also called in for consultation. Various adjustments are made, according to the nature and degree of impairment, from a simple change in seating to special tutoring. In some cases special training at the state school for the deaf is necessary, in order to prepare the child to return to the public schools later on. The program of conservation of hearing can be brought to a successful fulfilment only through the help of all persons in the community. The consultant in hearing and vision will collaborate with the superintendent of schools, the local health officer and the health education personnel, to arrange a plan of continued publicity and education designed to explain the value of hearing conservation to all members of the community.

Crowe and Burnam [32] again report on their investigative work concerning the diagnosis, treatment and prevention of hearing impairment

32. Crowe, S. J., and Burnam, C. F.: Recognition, Treatment and Prevention of Hearing Impairment in Children, Ann. Otol., Rhin. & Laryng. **50**:15 (March) 1941.

in children. During the past sixteen years, tests have been made on more than 15,000 patients, 3,000 of whom have been children. Their studies disclose that adenoids recur in more than 75 per cent of the children whose tonsils and adenoids were removed before the age of puberty. This recurrence frequently interferes with the function and ventilation of the eustachian tubes, producing an insidious painless type of progressive deafness. Complete blockage of the tubes causes subjective symptoms, but a partial or intermittent occlusion in children can be discovered only by the following signs and symptoms: impairment of hearing for the third or fourth highest tones—8,000 to 16,000 double vibrations; finding of hypertrophic lymphoid tissue in the nasopharynx, around the orifice of the eustachian tube, producing blockage, congestion of the mucous membranes of the middle ear and tube, hypersecretion of mucus and eventually fibrosis; retraction of either Shrapnell's membrane alone or the entire tympanic membrane, and, later, thickening of the tympanic membrane. If these signs are recognized early and treatment begun, hearing will be restored or further progress of the condition prevented in most instances. However, when fibrotic changes take place which permanently impair the mobility of the ossicular chain, no treatment can restore the hearing.

Examination of 1,365 Baltimore school children between the ages of 8 and 14 years revealed that only 58.8 per cent had normal hearing for all tones. Thirty-six and three-tenths per cent had impairment for the high tones with normal hearing for the low tones. The incidence of impairment was much higher in boys than in girls. Five per cent of the children had impairment of hearing for both high and low tones owing to chronic suppurative otitis media, otosclerosis and like causes. The incidence of high tone impairment was the same among those children who had been operated on for involvement of tonsils and adenoids as among those who had not submitted to operation. The reason for this is the great frequency of recurrence of lymphoid tissue in the nasopharynx of patients who were operated on.

The success of treatment depends on early recognition of the condition. Successful treatment is accomplished by a variety of measures: removal of enlarged tonsils and adenoids, followed by irradiation; occasional inflation of the tubes to remove thick tenacious mucus, and general hygienic measures. If the adenoids have recurred, irradiation rather than another operation is the proper treatment. The writers prefer the use of radium and describe the method which they employ and the applicator which they use. Roentgen treatment, however, can also be used, but special technic, larger doses and a longer period are required to accomplish similar results. The important points which the writers emphasize throughout their article are: the high tone type of deafness,

which is not a nerve or perceptive deafness; the necessity for early recognition; the good results which can be accomplished with proper therapy; the frequency with which lymphoid tissue recurs in the nasopharynx, around the orifices of eustachian tubes, and, finally, the necessity for periodic examination of the nasopharynx for the preservation of hearing.

Mao and Hughson [33] examined 331 children with profound hearing impairment requiring special education in the Pennsylvania School for the Deaf, to determine whether the eustachian tubes were patent within relatively normal limits. They used a method of measured autoinflation, taking 60 cm. of water as the maximum pressure required to open a normal tube. They found no apparent relationship between biologic deafness with profound hearing loss and inflatability of the middle ear. Thickening and retraction of the drum membrane were not particularly frequent in the patients whose tubes failed to inflate. Incidence of closed tubes apparently had no relation to age and sex. The patency of the tubes is not a factor of real importance in this type of profound deafness. In patients who were examined by the nasopharyngoscope, especially those of the older group, from 15 to 21 years of age, adenoid tissue near the eustachian orifice was an infrequent finding. Where it was found, it was not an important factor in relation to tubal patency. The method the authors describe for testing the patency of the eustachian tube is relatively simple. They suggest that the routine periodic use of this test for children with normal or slightly impaired hearing might serve as a warning of impending difficulty and as an index of the effectiveness of therapy. It should also aid in clarifying various uncertainties relative to the role which the eustachian tube plays in the development of chronic progressive deafness.

Hughson and co-workers [34] state that early nineteenth century reports of the training of deafened children with subsequent improvement in the hearing level are inaccurate, because there were no reliable test methods in use. There is no doubt that these handicapped pupils acquired, through training, certain elements of speech. Surveys of data derived from questionnaires were reported in detail in the Volta Review in 1937. These indicate an increase of about 60 per cent in the use of group hearing aids but are lacking in objective evidence as to the actual achievement in speech.

This study provides detailed statistical analysis of articulation ability in children taught by the auricular and oral methods. Auricular training

33. Mao, C. Y., and Hughson, W.: Patency of the Eustachian Tube in Relation to Profound Hearing Loss in Childhood, Arch. Otolaryng. **33**:824 (May) 1941.

34. Hughson, W.; Ciocco, A.; Witting, E. G., and Lawrence, P. S.: An Analysis of Speech Characteristics in Deafened Children with Observations on Training Methods, Laryngoscope **51**:868 (Sept.) 1941.

is limited to children who possess usable residual hearing. The use of accordions, flutes, horns and the like is superseded by the application of acoustic stimuli to deafened ears at accurately measured intensity levels and with control of quality as desired in the tone. Children were tested by means of an accurately constructed phonograph, with precautions taken against wearing of the surface of the record by the needle. A list of words was used, embodying the cardinal numbers, to provide the rate of pronunciation, plus words containing twelve different vowel sounds. Speech characteristics were graded in comparison with those of normal children of the same age and educational level. The speech characteristics studied were articulation, explosiveness and audibility of the first consonant, holding, explosiveness and quality of the vowel, explosiveness and audibility of the final consonant, syllable continuity and expression. These findings may be regarded as the first quantitative appraisal of the great strides made during the past fifty years in the habilitation of deaf children and of the direction in which further progress is needed. It still remains to overcome the deficiency noted relative to the voicing of vowels and the expression and articulation given words. Apparently more emphasis is needed on methods of training.

A marked superiority of children taught by the auricular method is noted in those elements which contribute most to the deviation of speech from the normal, viz., expression, articulation and quality of vowels. Almost 17 per cent of the children who were trained by the auricular method spoke with good expression, as compared with 1 per cent of those who were trained by the oral method. Speech characteristics are undoubtedly influenced by the amount of auditory perception. Children have been classified into three groups: (1) those who can perceive tones from 128 up to and including 2048 cycles; (2) those who hear only to 1024 cycles; (3) those who perceive no tones on the audiometer or at least only 128, 256 and 512. Children who perceive at least 2048 cycles have the best auditory acuity; those who perceive no tone higher than 512 have the worst.

Another factor which influences speech characteristics is the age at which hearing is lost. The later in life the child loses his hearing, the more speech he will have acquired. Children taught by the auricular method whose auditory acuity does not extend beyond 512 cycles and who became deaf at less than 2 years of age were superior in expression, articulation and quality of vowel to children in the oral group who acquired their deafness after 4½ years of age and who can perceive 2048 cycles.

The two groups have been subdivided into age classes—under 15 and over 15. The observations indicate that improvement of speech characteristics progresses in both groups, but that for the children trained by

the auricular method the progress is related to all speech elements, while for the children trained by the oral method the progress is not observed in expression and articulation of word and quality of vowel.

It is felt that the data reported here should fulfil a twofold purpose: 1. They should present a method of studying voice characteristics of deafened children. 2. They should show that regardless of age at onset, degree or duration of hearing loss, the auricular method of teaching is basically superior to the oral for all children with any residual hearing.

One further point, the expense for the education of children thus handicapped is relatively large. The handicap itself increases the number of years of training required before high school certificates can be given. If it is possible to lower this barrier in any way, much will have been accomplished. The use of the auricular method shortens the total period by approximately two years, resulting in Pennsylvania in a saving of $1,290 per child so trained.

Children with impaired hearing fall into three educational groups, as determined by the amount of loss, the age of the child at the time of the loss and such variables as environment, personality and intelligence. Elvena Miller [35] calls these three groups the hard of hearing, the partially deaf and the deaf. The hard of hearing are those having a hearing loss of 20 decibels in both ears. They require instruction in lip reading. The partially deaf are those who have enough hearing to develop some speech, even though it may be imperfect. These children require special training in language, lip reading and speech, as well as individual help with all other school subjects. They should be associated as much as possible with hearing children. As much of the work as possible should be taken with children of normal hearing. Group hearing aids are of great value to these children.

The third group, the deaf children, must be specially taught. Group hearing aids are of value in stimulating the fragment of hearing they may have and making it usable. It is important to start their training as early as possible, and some schools are starting that of deaf babies at 2½ to 3 years of age, taking advantage of the so-called babbling period. The younger the child when he enters a special school for the deaf, the better is the outlook for his developing normal speech and language. Several cities in the state of Washington have special schools for the hard of hearing as part of the public school system.

HEARING AND THE WAR

The physical standards governing the examination of registrants by the examining physicians of the United States Selective Service System are the same as those which have been developed by the United

35. Miller, E.: Education of the Acoustically Handicapped, Northwest Med. 39:369 (Oct.) 1940.

States Army Medical Corps for the examination of selected men at Army and Navy induction camps.[36] The general hearing requirement is that the registrant should have comparatively good hearing. The tests employed are the low conversational voice test and, when indicated, an audiometric test. The voice test is made simply by having one of the applicant's ears closed and approaching foot by foot until he hears the low conversational voice, not the whisper. The distance at which the voice is heard is designated by a fraction, the numerator being the distance in feet at which the voice is heard, as 20/20 when heard at 20 feet (6 meters), which is regarded as normal, and 10/20 when heard at 10 feet (3 meters). In case of doubt, the registrant should be blindfolded and a watch used instead of the voice. The ticking intensity of the watch used should be determined previously, and the distance at which the ticking is heard by a normal ear should be known. The audiometric examination, when used, will be conducted and recorded according to the method prescribed by the manufacturer of the instrument. The classifications are as follows:

In Class 1-A are *a*. Normal hearing. *b*. Hearing in each ear of 10/20 or better.

In Class 1-B are *a*. Hearing in one or both ears less than 10/20 but more than 5/20. *b*. Loss of one or both external ears, if the registrant has followed a useful vocation in civil life and the deformity is not too greatly disfiguring.

In Class 4 are defects such as *a*. Hearing less than the minimum hearing prescribed under class 1-B. *b*. Chronic purulent otitis media, with or without mastoiditis. *c*. Chronic perforation of membrana tympani.

Instructions are also provided for testing for malingering, with directions for Erhard's test and the Chimani-Moos test.

Macfarlan[37] wrote the editor of the *Archives of Otolaryngology* regarding the article "Physical Standards Under Selective Service," published in *The Journal of the American Medical Association* Nov. 2, 1940. This article refers to two tests for malingering, the Chimani-Moos test and the Erhard test. He describes these two tests in detail. The Chimani-Moos test employs the principle of lateralization when a tuning fork, namely, C-2, is placed on the median line of the vertex or against the incisor teeth. After the examiner has first determined which is the better hearing ear, by holding the fork at an equal distance from each ear, lateralization is ascertained. In sound-conducting impairments the sound is lateralized to the affected ear. A malingerer, however, not distinguishing any difference, will usually state that he hears the tone louder in the normal ear. If then the external meatus of the normal ear

36. Physical Standards Under Selective Service, J. A. M. A. **115**:1552 (Nov. 2) 1940.

37. Macfarlan, D.: The Physical Standards Under Selective Service, Arch. Otolaryng. **32**:1111 (Dec.) 1940.

is tightly closed, the subject, if really deaf, either will hear the tone better in that ear or may no longer be able to distinguish on which side he perceives the tone. A malingerer, with the so-called normal ear tightly closed, will say that he does not hear the tuning fork at all. The Erhard test is based on the fact that a normal ear, though tightly packed, is still able to conduct sound waves to a limited extent, as a loud watch tick heard at a distance of 2 or 3 meters. A malingerer, placed in the middle of a large room, closes the so-called deaf ear. A loud ticking watch is gradually brought toward the normal ear, and he is told to count the beats. The normal ear is then tightly closed and the so-called deaf ear examined. If the malingerer claims that he does not hear the watch tick at a distance of 1 or 2 meters, at which distance it should be heard by a closed normal ear, simulating should be suspected. Macfarlan criticized this test in that it deals entirely with malingering of hearing by the good ear and does not prove hearing in the ear claimed to be deaf. He also states that it seems strange that the Stenger test, which is the most familiar and efficient test, has been omitted by the military authorities. He would also like elucidation of the instructions as to how to determine when the use of the audiometer is indicated.

The Chimani-Moos test apparently does not take into consideration the lateralization of sound to the nonaffected ear in some cases of severe perception deafness.

Guild [38] discusses the various causes of deafness found during periods of war and the means of prevention. No cause of hearing impairment is peculiar to wartimes. However, the incidence is markedly greater in military personnel than in the corresponding age group of civilians in time of peace. Acoustic trauma due to exposure to recurrent extremely loud noise for long periods and detonation waves from nearby explosions is the type essentially dealt with in this article.

Other causes of deafness with increased incidence are epidemic spinal meningitis with complicating labyrinthitis, otitis media, wounds of the head, and rapid changes in altitude in combat flying and in training. The use of chemotherapy, the proper prophylactic care regarding infections of the upper respiratory tract and the wearing of steel helmets are means employed in an attempt to lessen the incidence of these conditions. No satisfactory means has been found as yet to prevent the serious injury to the hearing in flying due to rapid changes in altitude, producing tubal occlusion. Acoustic trauma due to the noise in flying has been conquered to some extent with the wearing of close-fitting ear caps or receivers of radio or intercommunicating apparatus, in addition to being enclosed in a cabin.

38. Guild, S. R.: War Deafness and Its Prevention, Ann. Otol., Rhin. & Laryng. **50**:70 (March) 1941.

Acoustic trauma is a danger not only to the military personnel but also to the civilian population. It causes a greater impairment of hearing for high tones than for low tones. In the milder cases, after the fatigue element has worn off, the impairment is limited entirely to frequencies above 2048 or 2896 cycles per second. Hearing for conversation is good except for the highest of the sibilant sounds, and high-pitched buzzing tinnitus is frequently complained of. The lesion is probably in the basal turn of the cochlea. There is no effective treatment for this condition. A simple detonation may cause permanent severe impairment of hearing for all tones with marked loss of usable hearing. The tympanic membrane is frequently torn by intense detonation waves but usually heals unless some infection supervenes. Investigations and observations during the first World War showed that rupture of the tympanic membrane tends to protect the inner ear. This was substantiated by animal experimentation.

To prevent damage by acoustic trauma, the sound has to be reduced. A finger tip placed firmly over the external auditory canal is sufficient. However, one cannot always anticipate the need, nor can the hands be available at all times. Solid obliterators keep out the sound but are unsatisfactory in that they do not enable the person to hear at all and also produce discomfort after they have been worn any length of time. Dry cotton, which is used frequently, is the least effective of all. Guild, in 1917-1918, made tests to determine the efficiency of many devices and methods. The device which proved most satisfactory was made by an English firm no longer in existence and was called the "scientific ear drum protector Tommy." This protector consisted of a hollow soft spherical rubber bulb with a short-necked opening on one side. The opening was surrounded by a soft rubber flange. This could be inserted easily, one size fitting almost all adult auditory canals without causing discomfort and permitting the usual voice sounds to be heard. It was very inexpensive. A footnote in this article states that the ordinary ear stopples give but little protection from detonation trauma. Guild concludes by stating that war deafness can be prevented only through the use of necessary technical information as well as suitable action taken by those in authority.

Craig [39] discusses injuries to the ear caused by explosion. The cause of injury is a violent displacement of air brought about by an explosive wave. The factor determining the injury is apparently the position of the external meatus in relation to the bursting bomb. There are three groups of injuries: (1) those affecting the tympanic membrane and middle ear: (2) those affecting the inner ear, and (3) hysterical deafness. In those

39. Craig, D. H.: Explosion Injuries of the Ear, Practitioner **145**:400 (Dec.) 1940.

of the first group there usually is hemorrhage in the substance of the drum membrane, and frequently a perforation. This type of injury is also associated with hearing impairment of the middle ear, conduction type. This is rarely permanent unless some severe secondary infection occurs and is neglected. Patients with injury to the inner ear often have a normal-appearing drum membrane. They complain of tinnitus and deafness, with a feeling of numbness around the ear. Usually, there is no complaint of pain or vertigo. The hearing tests show a loss greater by bone than by air conduction. In severe impairment there is loss of hearing for both ends of the auditory scale, high and low, and a greater loss for the high tones than in less severe impairment. Caloric response on the affected side is delayed. The prognosis in cases such as this should be guarded. The longer the deafness persists, the less chance there is for complete recovery. The caloric response may aid also in determining the prognosis. When the response is considerably delayed or unobtainable, serious damage to the inner ear may be inferred.

Hysterical deafness is complete bilateral deafness usually associated with other hysterical signs, such as blindness in one eye, paresis, hemianesthesia and mutism. The patients do not blink at the occurrence of a sudden noise, giving a negative cochleopalpebral reflex. They will not awaken if a loud noise is made beside them when they are asleep. They genuinely do not hear any sound. Therapy by a good psychologist should restore the hearing. The prognosis is good.

Davis [40] states that deafness may arise from injuries to the auricle and external meatus, to the drum membrane and middle ear or to the internal ear due to gunfire. Trauma to the auricle and external canal should be repaired, to prevent stricture and other unfortunate results, so that sound can still get through to the middle ear. Rupture of the drum membrane associated with bleeding, pain, deafness and vertigo is best treated by rest in bed for several days and the avoidance of all local interference. The drum membrane usually heals without any loss of hearing, in from five to ten weeks. There are three clinical types of injury to the internal ear arising from gunfire: (1) permanent structural damage to the internal ear; (2) psychic or functional damage, and (3) temporary deafness resulting from concussion. Reeducation and rest are the best forms of therapy for injuries of the latter two types. The exact pathologic changes associated with injuries of the first type are not known. Fraser, who made a microscopic study of the ears from 4 persons who died from injury of this type, found a hemorrhage at the fundus of the internal auditory meatus.

40. Davis, E. D. D.: Deafness Due to Gun Fire, M. Press **204**:33 (Oct. 30) 1940.

Wodak [41] discusses some of the conditions of the auditory apparatus which should be looked for in prospective pilots. Purulent otitis media, retraction of the drum membrane and chronic sclerosis of the middle ear with obstruction of the eustachian tube are conditions which should cause rejection of a candidate. Catarrh of the eustachian tube is also definite ground for rejection. A candidate with healed otitis and with an intact drum membrane or even a small perforation with good hearing need not be rejected provided the eustachian tubes are patent. At one time it was thought that orientation in the air depended on the vestibular apparatus, especially when vision was obstructed. However, recent work has shown that the proprioceptive sensibility is much more important in maintaining orientation than the mechanism of equilibrium in the inner ear. Candidates who have definite spontaneous nystagmus on looking straight ahead and those in whom the vestibular apparatus fails completely should be rejected.

Those with diminished responses, however, may be accepted. Hyperexcitability has been regarded by most investigators as sufficient ground for rejection. Some otologists attach less importance to the results of the tests than to the effect of a trial flight and suggest that the reactions to the latter be studied, especially those of the vegetative nervous system as indicated by nausea. The vestibular apparatus should be tested by modern methods and the results checked and recorded every six months, so that changes in the threshold can be observed. Further research in this field would be valuable.

Some years ago Wodak observed the development of physiologic hypoexcitability in pilots of long standing, showing that they were becoming accustomed to their profession. However, in spite of this, severe damage to the vestibular apparatus may occur in very stormy weather and pilots may suffer from severe air sickness, which might completely disorient them, leading to a catastrophe. The physiologic hypoexcitability of the vestibular apparatus is only temporary, and the condition becomes normal after a long period of rest.

The hearing is usually affected after exposure to noise. If a pilot experiences severe deafness after a comparatively short period of service, he should be sent on leave. If his hearing does not improve after a long period of leave or if it again becomes seriously impaired after he begins flying, the aviator should give up flying, in order to avoid greater and more lasting damage to his hearing. Ear plugs offer some protection. Pilots with acute head colds should not fly, because they are more susceptible than usual to variations in pressure and may endanger the safety of the machine and its occupants. Obstruction of the eustachian tubes is also likely to affect the pilot.

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41. Wodak, E.: The Auditory Apparatus and Aviation, Lancet **1**:8 (Jan. 4) 1941.

Dickson and Ewing [42] showed that there is a considerable variation in the amount of protection of the hearing provided by the best methods available at present. Their experiments employed both pure tone and noise tests. They found that the most effective devices tested were ear plugs of solid but pliable character which were capable of immediate adaptation to the size and shape of the individual meatus. A combination of effective ear plugs with the high altitude flying helmet prevented the elements of aural fatigue and tinnitus. Neither of these when used alone was completely successful in preventing aural fatigue. All of the methods of ear protection tested diminished the intelligibility with which speech could be heard. The test of interference approximated the degree of the efficiency of the protection. An analysis of airplane noise disclosed its transient and explosive character. This probably explains the high tone deafness caused by a predominantly low-pitched noise. Stevens and Davies state: "When we analyze a brief auditory stimulus, we find that the sudden beginning and the sudden termination of a tone are equivalent to the appearance of acoustic energy over a wide range of the spectrum." The first symptom of occupational or traumatic deafness is usually a loss of acuity for sounds of frequencies about 4096. This is probably due to the vulnerability of the basal turn of the cochlea to such sounds, because of its proximity to the oval window, which may expose it to the initial violence of thrusts of the stapes in response to impulsive sound. If the interpretation of the effect of airplane noise on the auditory acuity of flying and ground personnel is correct, there is hope that those persons may be effectively protected from the danger of deafness by the application of methods which exclude a great part of the noise, although low-pitched components cannot be wholly prevented from reaching the ear.

Perlman [43] discusses acoustic trauma in man. He mentions the following factors which influence the degree of hearing loss from acoustic trauma: the total time of exposure and the length of exposure in each period; lack of rest periods, conducive to greater trauma, and the loudness of the sound stimulus, loud sounds usually being the ones which cause damage to the ear. Age is a factor, older people being more susceptible than younger people and their auditory apparatus having less recuperative power. The character of the sound stimulus plays an important part. The middle ear muscles in man relax after about forty-five seconds of exposure to continuous sound, with the development of adaptation in the central nervous system. Sound stimuli repeated at slower intervals than one per minute or less or of brief

42. Dickson, E. D. D., and Ewing, A. W.: The Protection of Hearing, J. Laryng. & Otol. **56**:225 (July) 1941.

43. Perlman, H. B.: Acoustic Trauma in Man: Clinical and Experimental Studies, Arch. Otolaryng. **34**:429 (Sept.) 1941.

sound repeated more often will induce a complete cycle of contractions of the middle ear muscles. Each muscle contraction is preceded by a latent period, during which time the cochlea is unprotected. The fatigue of an ear thus unprotected causes a greater threshold change than that of a normal ear and necessitates a greater period for recovery. Loud sounds interrupted very frequently, such as those of rapidly repeating firearms, pneumatic tools and stamping machines, may have a much more injurious effect on the hearing than other sounds. Sounds of very sharp onset, such as those from explosions and firearms, are due to single exceedingly brief compression impulses of enormous magnitude and pressure. These affect the high audible frequencies, and only a single exposure to such a sound may cause a permanent loss of hearing for high frequencies. Other factors, such as constitutional changes, the use of protective devices, as helmets or cotton plugs, exposure in an enclosed or open space and previous aural disease, may also influence the degree of hearing loss from acoustic trauma. Perlman's investigation deals mainly with the relationship between the frequency of the stimulus and the resultant audiogram. The greatest depression, however, is not at the frequency of the fatiguing tone but usually one octave above. This was true for 512, 1024, 2048 and 4096 cycles. The results at 256 cycles were not uniform, and tests could not be made for 8192 cycles. A uniform threshold curve, namely, with a 4096 cycle dip, was not obtained for a series of tones of approximately uniform intensity used to produce acoustic trauma. The curve normally found in early acoustic trauma with the 4096 cycle dip was obtained in subjects exposed to a fatiguing tone of 2048 cycles. Sounds of low frequency produced less acoustic trauma than those of higher frequency of approximately equal intensity. A fatiguing tone of 4096 cycles produced a localized drop at 8192 cycles but not at 4096 cycles. The upper tone limit was not affected by any fatiguing tone used. Petrolatum-treated cotton provided complete protection against the stimulus used at all cycles except 1024, at which only partial but definite protection was effected. Tinnitus following exposure was of variable duration and though not regularly identified with the frequency of the fatiguing tone was often an octave above it. It was increased in intensity by closure of the external auditory canal and when stopped could be made to reappear by closing the canal with the finger. The tinnitus usually stopped before the threshold curve became normal, thus showing that the values of the threshold determination were hardly interfered with by the tinnitus.

Berendes [44] states that the audiogram indicating temporary nerve deafness following sudden noise, such as pistol shots, is misleading. The

44. Berendes, J.: Audiogram of Transitory Deafness Due to Noise and Its Interpretation, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **147**:281, 1940.

function of the ossicles must be considered, at least when a high-pitched tinnitus is present. If one prepares for the noise by closing the eyelids forcibly, the tensor tympani is tensed and acts as a preventive.

Bekesy [45] measures both the pressure and the intensity of industrial noises transmitted not only by air or bone conduction of the head but by vibrations perceived through the feet and other contact points of the body. With frequencies up to 10,000, sounds may be carried through the head without damping by the skin. The hearing curve on sounds carried by contact with the teeth is similar. The vibrations in the cochlea are identical by air and bone conduction.

45. Bekesy, J.: The Diffusion of Sound by Bone Conduction, Ztschr. f. Hals-, Nasen- u. Ohrenh. **47**:430, 1941.

ANATOMY AND PHYSIOLOGY OF THE EAR

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In reviewing the literature from January 1940 to November 1941 pertaining to the anatomy and physiology of the ear, we present summaries and abstracts of articles which permit the reader in a brief time to obtain a panorama of the field concerned. This review is not intended to be self sufficient, as are the original papers. Only articles of special value to us are included in this review.

DEVELOPMENTAL ANATOMY

In a continuation of his studies of the development of the otic capsule, Bast [1] discusses residual cartilages and defective ossification and their relation to otosclerotic foci. He observed that there are six regions of the petrous bone where there is residual cartilage or defective bone: (1) that of the fissula ante fenestram, commonly; (2) that of the fossula post fenestram, rarely; (3) the infracochlear region; (4) the base of the styloid bone; (5) the region of the petrosquamous suture and the capsule beneath the suture, and (6) the region of the semicircular canals. In discussing these areas the author speculates that it may be an attempted repair of such bony defects through maturity that leads to the formation of otosclerosis. His customary excellent photomicrographs were included to illustrate his paper.

Perlman [2] describes a clearly defined small triangular crescent-shaped area of reenforcement of the saccular wall. This is present in man but absent in the lower mammalian forms. This reenforcement is due to connective tissue entering from the margins of the vestibule and separating the epithelial and the mesothelial layer of the saccular wall. The author thinks that this area has some functional significance and is not merely a support for blood vessels.

Tenaglia [3] states that it becomes more difficult to study the otolithic apparatus in the higher animals, because of the necessity of abolishing

1. Bast, T. H.: Development of the Otic Capsule, Arch. Otolaryng. **32**:771-782 (Oct.) 1940.

2. Perlman, H. B.: The Saccule, Arch. Otolaryng. **32**:678-691 (Oct.) 1940.

3. Tenaglia, G.: Function of the Otolithic Organs, Oto-rhino-laryng. internat. **23**:573 (Nov.) 1939.

other functions that have been grafted on it. In studying the otolithic organs, the method of McNally and Tait of severing the individual vestibular branches in the frog, is the most rational. The otolith has high specific gravity, and otolithic reflexes in response to a stimulus are due principally to the force of gravity. Recently, it has been pointed out that compensatory ocular movements are dependent on the utricle, since they persist after destruction of the sacculus. Likewise, it has been shown that the righting reflexes are localized in the utricle. The reflexes of static adaptation and the tonic labyrinthine reflexes which maintain equilibrium are also of utricular origin. To each pair of otoliths a special function can be attributed. The lapilli are said to control the flexor and extensor muscles of the neck, trunk and limbs and vertical movements of the eyes. The sagittae control rotatory ocular movements and the adductor and abductor muscles. However, recent studies exclude the saccule from participation in the maintenance of equilibrium.

The anatomy of the salpingopharyngeus muscle is taken up in detail by McMyn.[4] He presents some excellent diagrams of dissections of this rather neglected muscle. He believes that the eustachian tube is opened chiefly by the tensor palati muscle. The levator palati, the salpingopharyngeus and the fibers of the superior constrictor muscle may act synergically in opening the tube. Closing is probably passive and is aided by the elasticity of the cartilage. The author finds that normally the walls are very close or else in contact. This holds true for the region between the isthmus and the midportion of the cartilaginous tube. In the pharyngeal orifice the walls are open. The tube varies individually. In some cases it is always open, and in others mucus has closed it.

The eustachian tube has been studied histologically by Polvogt and Babb.[5] They emphasize the number of glands in the cartilaginous portion and the varying degree of vascularity of the tunica propria. They found lymphoid tissue in all parts of the eustachian tube. It is found commonly in the pharyngeal end. Of course it is most prominent in young persons, but the authors found lymphoid tissue in persons of 65. This study dealt only with the eustachian tubes of subjects with normal hearing.

Lierle and Potter[6] have written an excellent paper on the middle ear from the standpoints of physiology and histopathology. There are thirty-six photomicrographs. This paper should be studied by all who are interested in the subject.

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4. McMyn, J. K.: The Anatomy of the Salpingo-Pharyngeus Muscle, J. Laryng. & Otol. **55**:1-22 (Jan.) 1940.

5. Polvogt, L., and Babb, D. C.: Histological Studies of the Eustachian Tube of Individuals with Good Hearing, Laryngoscope **50**:671-675 (July) 1940.

6. Lierle, D. M., and Potter, J. J.: Physiology and Histopathology of the Middle Ear, Ann. Otol., Rhin. & Laryng. **50**:235-270 (March) 1941.

In an attempt to determine whether there is any change within the membranous labyrinth due to a change in intracranial pressure, Fischer [7] selected 5 cases in which the patient had died of neoplasm of the anterior and middle fossa, having had no surgical treatment. Ten petrous bones were serially sectioned for examination. Detailed case reports and photomicrographs illustrate the paper. Changes in the inner ear caused by chronic increased brain pressure are classified as follows: (1) transudation, exudation and lymphatic congestion (dropsy and edematous infiltration) within the membranous inner ear and the nerve ganglion apparatus (vaginal dropsy of the seventh, eighth and ninth nerves); (2) hyperemia (especially venous), hemorrhages and alterations of the walls of the blood vessels within the inner ear and the petrous bone; (3) formative changes within the membranous inner ear (ectasia, stenosis and collapse) and in the ductus cochlearis, the ductus endolymphaticus, the ductus perilymphaticus and the ductus reuniens; (4) formation of hernias of the brain and pacchionian granulations; (5) secondary (later) changes, such as pigmentations, attachments and adhesions in the membranous inner ear and ascending atrophy of the nerve ganglion apparatus; (6) pressure atrophy of the bone due to osteoclasts and Volkmann's perforating canals, caused by herniations of the brain and pacchionian granulations. The author finds an analogy between the choked disk of the eye and the congestive inner ear. The pathways by which brain pressure is transmitted into the inner ear are the subarachnoid spaces of the aquaeductus cochleae, the nerve sheaths in the internal auditory meatus, the pacchionian granulations, the ligamentum spirale cochleae, the lymph channels of the bony labyrinthine capsule and the blood vessels. Increased generalized brain pressure may be present over a certain period without damaging the delicate sensory epithelium and the nerve ganglion apparatus, even when extensive transudation or exudation is present. When such pressure has been active over a long period it produces secondary degenerative processes extending from the base of the cochlea to the top. It is the duty of the otoneurologist to discover by exact and periodically repeated examinations a congestive condition of the inner ear in the earliest stage and to point out the indications for surgical intervention before irreparable changes occur in the delicate sensory apparatus.

Rasmussen [8] counted the nerve fibers in 37 normal vestibular nerves and 40 normal cochlear nerves from persons with apparently normal

7. Fischer, J.: Changes in the Internal Ear Due to Increased Endocranial Pressure, Arch. Otolaryng. **31**:391-419 (March) 1940.

8. Rasmussen, A. T.: Studies of the Eighth Cranial Nerve of Man, Laryngoscope **50**:67-82 (Jan.) 1940.

hearing whose ages ranged from 2 to 60 years. Normal cochlear nerves contain from 22,800 to 40,000 nerve fibers, the average being 31,400. These figures are distinctly higher than those of any fiber counts on human material previously discovered in the literature. The close agreement between these data and the independent figures on the number of spiral ganglion cells makes it safe to regard the number of conductors carrying auditory impulses into the human brain under normal conditions as about 23,000 to 40,000. The average number of cochlear fibers in man appears to be between 31,000 and 32,000, compared with nearly 50,000 in a cat. The cochlear nerves of the older subjects contained 2,200 fewer fibers and the vestibular nerves 1,000 fewer fibers than those of the younger subjects.

PHYSIOLOGY OF HEARING

The physiology of hearing as it is understood today is well described by Davis.[9] The sensation of pitch is determined by the position of the vibratory pattern on the basal membrane. A map or graph of the basilar membrane is constructed on the basis of six different sets of data: (1) the position of maximum electrical activity; (2) impairment of electrical activity by localized lesions; (3) pathologic changes associated with high tone deafness; (4) integration of just noticeable differences of pitch; (5) the contribution of different parts of the basilar membrane to the total loudness of a sound, and (6) the relation of pitch, subjectively perceived, to the frequency of a tone. These different sets of data agree amazingly. The graph or map establishes the low tones at the helicotrema, frequency 2200 at the midpoint and the high tones at the start of the first turn, close to the oval window. Thus, the analyzing mechanism of the inner ear is the essential part of the mechanism of hearing. In order that a tone may be heard, nerve fibers that innervate a part of the basilar membrane must be stimulated.

Kobrak, Lindsay and Perlman[10] name as the most desirable "next step in auditory research" a better understanding of the cochlea and its mechanical functioning. They then describe experimental possibilities for the study of the function of the cochlea. Using cats, they introduced a glass tube into the vicinity of the round window. The tube was held in place by wax. It was demonstrated that sound may enter the cochlea through the round window and reach the nerve endings through the scala tympani and not through the scala vestibuli. An attempt was made to conduct a similar experiment in relation to the oval window, but this

9. Davis, H.: The Clarification of Certain Phases of the Physiology of Hearing, Laryngoscope **50**:747-755 (Aug.) 1940.

10. Kobrak, H. G.; Lindsay, J. R., and Perlman, H. B.: The Next Step in Auditory Research, Arch. Otolaryng. **31**:467-477 (March) 1940.

was difficult and the results were not clear. The test was carried out by placing the tube in the external auditory meatus, and the cochlea function thus determined was compared with that obtained by testing through the round window. To determine whether sounds could go into the cochlea without passing through the oval window, the stapes was destroyed or extirpated. It was found that bone conduction is possible without the stapes. The round and oval windows were then closed over with plaster of paris. It was found that when one window only was made rigid, bone conduction was unchanged or even improved, but when the regions of both windows were made rigid, function could not be demonstrated by either bone or air conduction. When the labyrinthine fluid was removed by tapping the cochlea, it was observed that it was impossible to transmit sound through the lamina spiralis ossea. It was thus evident that the labyrinthine fluid must be present in order to transform sound from air to nerve impulses. The authors then considered the internal auditory meatus, the blood vessels, lymph spaces and aquaeductus cochleae as a third labyrinthine window and called it the "craniolabyrinthine communication." By a sensitive recording mechanism they demonstrated that very small pressure changes in the cranial cavity produce movements in the labyrinthine fluid. It was found that relatively large increases in pressure with slow oscillatory movements can be demonstrated in the cochlea, whereas pressures with rapid oscillations did not record.

Wever, Bray and Lawrence [11] performed experiments to determine the cochlear activity after death. Their experiments were conducted on guinea pigs and cats. The potentials were picked up by a silver foil electrode on the round window membrane and after amplification were measured either with a cathode ray oscillograph or a wave analyzer. The stimuli were pure tones from a loud-speaker. Death was caused by (1) pithing the medulla, (2) clamping the heart, (3) clamping the trachea and (4) poisoning with curare. A prompt impairment of response was obtained by all these procedures, but that obtained by clamping the heart was the most prompt. Response to the high tones showed the most rapid decline on the whole, but this was not constant. After a large initial loss, the responses declined relatively slowly, and responses were still present after five hours but not after twenty-four. It was suggested that the cochlear potentials arise through a direct translation of stimulus energy and not through a liberation of stored energy. These experiments were carefully controlled, to rule out experimental errors.

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11. Wever, E. G.; Bray, C. W., and Lawrence, M.: The Nature of the Cochlear Activity After Death, Ann. Otol., Rhin. & Laryng. **50**:317 (June) 1941.

With the advent of the fenestration operation, hearing tests are receiving greater attention and study. In the office one may with a short audiometric examination determine whether or not a hearing aid is indicated. Tuning fork tests alone are sufficient for a routine diagnosis. But F. W. Kobrak [12] suggests that in all cases of special scientific interest both tuning fork and audiometric tests are indicated. He describes several new hearing tests and suggests that in the case of tuning forks a series test is of value. He uses Struycken's tuning forks. These tuning forks are calibrated according to the period of perceptible vibration time. It has long been known that when a fading tuning fork has ceased sounding but not vibrating the patient can hear the sound again when the fork is brought back to the ear after a short interval. Kobrak makes a good deal of this observation and measures the several threshold values which can be obtained by this procedure. Curves plotted for these sounds may be of value in determining types of deafness. Kobrak also discusses audiometric results based on crescendo and diminuendo readings. A crescendo reading is taken at the point at which the patient first hears a tone when the intensity is increased slowly, while a diminuendo reading is taken at that point at which the patient fails to hear the audiometric tone when the intensity is decreased. The author also discusses the bone conduction audiogram. He thinks that a dip at the frequency 1024, while indicating an impairment in hearing, does not always mean cochlear damage. It is found in cases of middle ear deafness, but in these cases the cochlea may well be affected. Early lesions at the 1024 dip are probably due to a weak spot in the cochlea.

The Rinne test is widely accepted in clinical otologic practice. Experience has shown that it helps to localize a lesion in the middle or in the inner ear. Bunch [13] compares the results of the Rinne test made with a tuning fork with those obtained by the use of an audiometer. He compares the audiometric thresholds of air and bone conduction for the tone 512 with those obtained with a C-2 weighted tuning fork. In most cases in which there is a difference of more than 45 decibels between the air and the bone conduction threshold, the Rinne test is positive. When the difference is less than 45 decibels, the Rinne test is negative. When the difference is 45 decibels (plus or minus 5 decibels), the Rinne test will be "indeterminate" or "neutral." This range of threshold difference affords opportunity for the study of its clinical significance. By the use of the audiometer two factors of error in the Rinne test can be

12. Kobrak, F. W.: New Tests and Clinical Experiments on Hearing, J. Laryng. & Otol. **55**:405-423 (Sept.) 1940.

13. Bunch, C. C.: The Rinne Test and the Audiometer, Ann. Otol., Rhin. & Laryng. **50**:47-54 (March) 1941.

eliminated. First, the error due to the constant decrement of the tuning fork is eliminated, because the intensity of the audiometric tone for both air and bone conduction remains fixed at each setting. Second, errors due to possible variations in pressure and in selection of the point of mastoid contact are eliminated, because the audiometric tonal intensity is maintained without change.

Luscher [14] carried out experiments producing disturbances in the sound conduction apparatus and then tested the patient's ability to hear. The subjects used had normal hearing. When he created a middle ear or air conduction deafness, by various methods, he observed that there was a loss for the middle and high tones. By placing on the umbo a a metallic rod, introduced through a speculum and held to the tympanic membrane with a solution of mastic in benzene (mastisol), he could add weights up to 2 or 3 Gm. The result of this was a loss for the middle and low tones and a normal perception for the high tones. The upper limit was normal. This is a rather surprising finding. Second, 0.5 to 2 Gm. of mercury was placed in the auditory canal. The drops first covered the inferior anterior quadrant of the drum membrane and then covered the pars tensa. In testing the hearing, the author found that the audiometric curve was lowered and that the upper limit had dropped. The acuity for the middle and high tones was particularly affected. The larger the area covered by the mercury, the greater the loss of hearing. Third, filling the meatus with water produced the greatest lowering of the audiometric curve. The upper limit was especially low. There was a loss in hearing for all tones. That for middle and high tones was the greatest, the maximum being at 4096. These artificial disturbances parallel pathologic occlusions of the meatus. Loading of the drum, the author says, is comparable to occlusion of the eustachian tube. He believes that adhesions and otosclerosis, both affecting the chain of ossicles, are similar in their effect to loading of the umbo with a metallic rod. In pathologic conditions the disturbances of conduction are not uniform in a mechanical or an acoustic sense. Hitherto a disturbance in the conductive apparatus has been thought to cause a loss of hearing for the low tones. The author's experimental work seems to indicate that a loss of hearing for high tones may well result.

Nor is clinical evidence lacking. Crowe and Burnam [15] stress their contention that impaired hearing for high tones with good hearing for low tones is the earliest symptom of middle ear as well as of inner ear

14. Luscher, E.: The Effect of Experimental Disturbances of Tympanic Membrane on Hearing Capacity, Acta oto-laryng. **27**:250-267, 1940.

15. Crowe, S. J., and Burnam, C.: Recognition, Treatment and Prevention of Hearing Impairment in Children, Ann. Otol., Rhin. & Laryng. **50**:15-31 (March) 1941.

deafness in children. They state that the recurrence of adenoid tissue after operation in young children is so common that it must be regarded as normal. This recurrent lymphoid tissue may impair the function of the eustachian tube. For good hearing the eustachian tube must function normally. Recurrent nodules of adenoid tissue in and around the pharyngeal orifice of the eustachian tube cannot be removed surgically. This lymphoid tissue is so sensitive to radiation that a very small dose of radon reduces its size without harm to any of the surrounding tissue. The sole purpose of irradiation is to reduce the obstructive nodule of lymphoid tissue and restore normal ventilation to the eustachian tube. The younger the patient with this difficulty, the fewer the sequelae in the middle ear and consequently the better the result of the irradiation. The authors emphasize the importance of early recognition of impairment of hearing for high tones and the use of the nasopharyngoscope. Their method successfully attacks the problem of the prevention of progressive deafness due to blocking of the eustachian tube.

During the year 1939-1940, Guild and his associates [16] carefully examined 1,365 Baltimore school children from 8 to 14 years old. So far in this study, which is still in progress, two important factors have been established. First, more than 40 per cent of the children did not hear all the tones in the range of intensity commonly regarded as normal. Secondly, three fourths of them had nasopharyngeal conditions regarded by the examiners as a potential menace to hearing. The authors present charts demonstrating the loss of hearing which is common for the high tones by correlating the hearing loss with the age and sex of the patient, the season of the year and the amount of lymphoid tissue in the region about the eustachian tube. Radon therapy is being used to shrink the lymphoid tissue. A subsequent report should reveal whether radon therapy is as effective as surgical intervention in preventing the regrowth of adenoid tissue. If this group of children is representative of the public at large, the prevention of impairment of hearing in children is a much greater problem than has been generally appreciated.

Mendel's law applies to the inheritance of night blindness, hemophilia and other afflictions. Ersner and Saltzman [17] report 3 cases of familial deafness involving three successive generations. Fifty per cent of the females of three successive generations had analogous nerve types of deafness. In each instance the right ear was the first affected. These cases seem to illustrate the mendelian law and to show a sex-limited inheritance.

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16. Guild, S. R., and others: Impaired Hearing in School Children, Laryngoscope **50**:731-746 (Aug.) 1940.

17. Ersner, M. S., and Saltzman, M.: Progressive Analogous Nerve Deafness in Three Successive Generations with Sex-Limited Inheritance, Laryngoscope **51**:241-245 (March) 1941.

The embryologic develpoment of the ear is suggested by Jones [18] as a guide to vitamin therapy. He presents a thorough review of the literature on vitamins relevant to the eye, ear, nose and throat. He emphasizes that each vitamin has a specific relationship to one of the three primary germinal layers: ectoderm, mesoderm and entoderm. While all the layers may be affected, each vitamin has a preponderant effect on one layer. Vitamin A and vitamin B complex affect the ectoderm chiefly. Vitamin C and vitamin D affect the mesoderm chiefly. Only when there is a gross deficiency is it easy to demonstrate a lack of vitamins. Advanced deficiency states are rare. The large number of patients with a moderate or mild deficiency are hard to identify. Unless a patient has such a deficiency, a cure cannot be expected from vitamin therapy. According to Jones, if too large an amount of vitamins, with the exception of D and B complex, is taken, no harm is done to the patient. He believes that the administration of vitamins is best accomplished by subcutaneous injection.

Covell [19] holds that treatment with vitamins must still be regarded as in the experimental stage. From work on experimental animals it is seen that there is a correlation between diseases of the internal and middle ear and a lack of vitamins in the diet.

Vitamin therapy for deafness in human beings has been limited largely to the use of vitamin B complex. When the hearing impairment is related to a dietary deficiency, improvement with vitamin therapy may be expected. Covell,[20] who has demonstrated degeneration of the cochlear nerve in various vitamin deficiencies, has extended his studies much further. He reports in detail histologic studies of the changes that are specifically due to various vitamin deficiencies. The author's summary follows:

1. Vitamin A deficiency in rats is associated with middle ear infection, increase in the periosteal layer of the otic capsule and occasionally exostoses in the internal auditory canal, degeneration of the cells of the stria vascularis, external sulcus cells and parts of the organ of Corti, with slight to moderate demyelinization of the cochlear nerve.

2. Vitamin B_1 and various factors of the vitamin B_2 complex result in a higher incidence of middle ear infections, when one of them is lacking in the diet of animals. Avitaminoses of the B group cause no significant changes in the bone of the otic capsule or ossicular chain. Degenerative changes are found in the external hair cells of riboflavin deficient animals. Spiral ganglion cells are altered

18. Jones, I. H.: Vitamins and the Eye, Ear, Nose and Throat, Laryngoscope **50**:585-627 (July) 1940; Vitamins and the Ear, Nose and Throat, ibid. **51**:609-669 (July) 1941.

19. Covell, W. P.: Vitamins and the Ear, Laryngoscope **51**:683-691 (July) 1941.

20. Covell, W. P.: Pathologic Changes in the Peripheral Auditory Mechanism Due to Avitaminoses (A, B Complex, C, D and E), Laryngoscope **50**:632-647 (July) 1940.

in appearance in all avitaminoses of the B group, less for B_6. Demyelinization of the cochlear nerve, particularly in the dendritic portion, has been found to be more severe in animals maintained on these diets. In fact, the anti-gray hair factor is the only exception. According to the present series the three most important vitamins for the maintenance of the myelin sheath of the nerve are, in order, vitamin B_6, thiamin chloride and riboflavin. Such evidence as we have to date indicates that the cochlear nerve is more susceptible to alterations than the vestibular portion of the eighth nerve or any other cranial nerve, with the possible exception of the optic. An explanation may be found on the basis of phylogeny, because the cochlear nerve is relatively recent phylogenetically.

3. Vitamin C deficient guinea pigs revealed hemorrhage into the lining of the middle ear cavity with an increase in the thickness of the submucosa. There is a tendency for cellular marrow to be replaced by fibrous marrow. Only slight demyelinization of the cochlear nerve is produced. The cells of the organ of Corti, including the hair cells, are swollen and degenerated, and also some of the cells of the stria vascularis. Chronically deficient vitamin C guinea pigs may reveal more extensive changes, particularly within the ossicular joints and the bone of the capsule. This subject is to be further studied.

4. Vitamin D deficient rats frequently have infections of the middle ear. This deficiency is to be associated with the laying down of osteoid in the bone of the ossicular chain and otic capsule. Demyelinization of the cochlear nerve also accompanies this vitamin lack. The effects of hypervitaminosis D_2 on the bone of the capsule of young animals is to be reported subsequently.

5. Vitamin E low rats (third generation) show middle ear infections. The muscles of the middle ear, particularly the tensor tympani, contain considerable fibrous tissue replacement of the muscle bundles. It has been found for other deficiencies that the presence of an otitis media may result in atrophic changes within these muscles, because of the penetration of the exudate into the muscle substance and subsequent inflammatory reaction. Small exostoses and irregularities in the periosteal bone are in evidence. Hemorrhages about the nerve in the modiolus, under the dura and submucosa of the middle ear are present in these "E" deficient animals.

Vitamin A deficiency produced experimentally in rabbits is similar to that produced in rats in some respects and differs in others. Perlman and Willard [21] fed rabbits on vitamin A deficient diets and studied the cochlear function, the vestibular function and the histopathologic changes. It was found that there developed an extensive hypertrophy of the periosteal layer of the otic capsule on the cranial aspect, including the internal acoustic meatus, posterior to the fossa surface and mouth of the flocculus. The overgrowth of mature lamellar periosteal bone in the internal acoustic meatus resulted in narrowing and elongation of the internal acoustic meatus, with compressing and stretching of the nerve and of Scarpa's ganglion in the meatus. In spite of this compression and stretching good cochlear function was retained. This was demonstrated by threshold studies on the acoustic middle ear muscle reflex. The compression and stretching of the nerve resulted in various degrees of degen-

21. Perlman, H. B., and Willard, J.: The Ear in Experimental Vitamin A Deficiency, Ann. Otol., Rhin. & Laryng. **50**:349 (June) 1941.

erative change in some of the cells in the vestibular and cochlear ganglion, and in a single case in degeneration of the peripheral fibers to part of the basal coil. There was no evidence of serous labyrinthitis or of degeneration of Corti's organ, the vestibular end organs or the stria vascularis. The structures of the middle ear, including the adjacent labyrinth bone, were unaffected.

Long-continued use of vitamin B in cases of nerve deafness is recommended by Guggenheim.[22] When the nerve elements are destroyed, no treatment is worth while. But when the nerve deafness is secondary to focal infection, removal of the focus alone may not be sufficient. Intensive therapy with vitamin B complex should be tried. The author believes that in otosclerosis there is not only an osseous dystrophy but also a neuropathy. Vitamin B therapy is of value during pregnancy. The author states that during this time, even when otosclerosis is not present, the requirement of vitamin B is five times the amount needed normally. In acute infectious diseases the systemic need for vitamin B is greater than the normal requirement. In such diseases as mumps, measles, scarlet fever and typhoid, vitamin B may act to prevent involvement of the eighth nerve. Three or four thousand units of thiamine and 100 mg. of nicotinic acid are necessary daily, in addition to other vitamins. Again for the initial active stage of Ménière's disease the author recommends five times these doses, to prevent neurodegeneration. The result of such doses of vitamin B is in certain cases to improve the hearing and in all cases to improve the general health. The author states that when structural changes are present the treatment must be persisted in for eighteen months or more.

Selfridge [23] claims that estrogens probably play an important part in conduction deafness. He states that there is definite evidence that the thyroid and pituitary glands and the adrenal cortex are important in the treatment of this type of deafness. The vitamins, mineral salts and amino acids are related factors. Slowly the evidence is accumulated that nutritional deficiencies at the time of gestation cause deafness. Prevention of deafness therefore depends on the mother's getting an optimum diet and proper endocrine therapy during pregnancy. These dietary habits must be carried on during life.

The problem of the physiology and repair of an artificial bony fistula continues to bring forth articles by which an endeavor is made to shed new light on the subject. Guggenheim and Guggenheim,[24] using the

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22. Guggenheim, L. K.: Treatment of Deafness, Laryngoscope **50**:773-783 (Aug.) 1940.

23. Selfridge, G.: Chronic Progressive Deafness with Special Reference to Estrogenic Substances (A Further Contribution), Ann. Otol., Rhin. & Laryng. **49**:53-75 (March) 1940.

24. Guggenheim, L. K., and Guggenheim, P.: Artificial Fistula in Cases of Otosclerosis, Arch. Otolaryng. **32**:1-15 (July) 1940.

skulls of white rabbits as their experimental mediums, created holes with a sharp chisel and with a burnishing burr. Two months after the operation they studied their preparations microscopically and found that the fistulas created with a sharp instrument did not close with the formation of new bone as readily as those created with a burnishing burr. They maintain that the fact that the labyrinthine capsule consists of both membrane and cartilage bone in no way affects the results of the experiment, as all new bone formation, whether in the labyrinthine capsule or elsewhere, is by a membrane-bone process. They also emphasize the necessity of examining sections throughout each fistula, in order to obtain a clear picture of the region as a whole. They believe, as many others do, that in the final analysis of osteogenesis the individual characteristics of primary germinal tissue (mesenchyme) will doubtless prove to be the determining factor, just as they are the determining factor in such conditions as chondrodystrophy and osteogenesis imperfecta.

Lempert,[25] in an excellent paper, gives the indications and technic for endaural fenestration of the horizontal semicircular canal in otosclerosis. He holds that surgical intervention for the improvement of hearing in otosclerosis is no longer in the experimental stage. There is no risk to life. Postoperative discomfort is slight. In his experience physical hearing can be fully recovered in 80 per cent of carefully chosen cases. The hearing improvement following fenestration is the result of mobilization of the perilymph and endolymph by air-borne sound. Before operation this mobilization is prevented by the fixation of the stapes in the oval window. The newly created fistula replaces the damaged oval window. For best results it is necessary that the tympanic air space be sealed by the tympanic portion of the tympanomeatal membrane and that the eustachian tube, which regulates the air pressure in this tympanic air space, be patent. The sole problem is that of the best method of creating a permanent fenestra. Lempert describes in detail his method of making a permanently sealed fistula.

Asherson [26] explains nerve deafness on a vascular basis. He assumes that the cochlear nerve is a composite nerve, made up of at least two main branches. One portion, the more vulnerable, is concerned with the transmission of high tones; the other, less vulnerable, is concerned with the transmission of low tones. The second portion is affected only after the high tone branch has been involved, never before. He records a case of an unusual polyneuritis, a form of herpes zoster oticus. The

25. Lempert, J.: Endaural Fenestration of the Horizontal Semicircular Canal for Otosclerosis: Indications, Technique, Observations as to Early and Late Post-Operative Results, Laryngoscope **51**:330-362 (April) 1941.

26. Asherson, N.: The Cochlear Nerve and the Vascular Theory of Nerve Deafness, J. Laryng. & Otol. **55**:531-545 (Dec.) 1940.

paralysis involved the face as well as the vestibular and cochlear portions of the eighth nerve. The paralysis was transient, and the patient recovered completely. Asherson carefully followed the patient's hearing with audiograms. He believes that the patient's symptoms were due to a vascular occlusion. The recovery of the patient was due to complete and early restoration of the blood supply to the nerve. Asherson feels that the loss of high tones in previously accepted cases of nerve deafness is explained in the same way. For example, loss of hearing in the 4096 frequency is explained by assuming that noise above a certain intensity produces a spasm of an end artery distributed to part of the high tone division of the cochlear nerve which transmits the frequency 4096. With persistent exposure to the stimulus, the remaining part of the vessels to the high tone division of the cochlear nerve is occluded by spasm. This deafness will be permanent for that tone when the exposure is continuous. Or the deafness may be temporary and disappear when the removal of the stimulus soon enough leads to opening of the vessel and restoration of nerve function.

With war spreading over the world today, the protection of hearing assumes great importance. Exposure to the sound of the explosion of a single shell or mine may result in chronic deafness. Exposure to loud sounds, if long continued, results in chronic deafness. It has been observed that when one is subjected to loud sounds there is a complaint of tinnitus. When tinnitus results from daily or nightly exposure, it constitutes a warning sign that chronic deafness may ensue. Dickson and Ewing [27] give an account of their experiments to ascertain how effective are the devices at present available for the protection of the sense of hearing. In order to eliminate the risk of deafness, occlusion of the external auditory meatus or complete covering of the external ears is necessary. A combination of these two methods gives the best result. But when devices for protection from the loudness of external sounds are employed, the ability to hear speech is diminished. An exception to this is provided by the helmet worn by high altitude flyers, in which ear phones form part of the protection. High tone deafness caused by airplane noise especially was studied. Airplane noise, the authors state, is predominantly low pitched. They emphasize that the component parts of airplane noise are impulsive and explosive in character. This may mean that the basal turn of the cochlea, because of its proximity to the oval window, is damaged by the initial violence of the thrusts of the stapes in response to these impulsive and explosive sounds. Thus, for airplane pilots, protection from the danger of deafness can be attained only by the skilled application of protective devices that insulate the

27. Dickson, E. D., and Ewing, A. W. G.: The Protection of Hearing, J. Laryng. & Otol. **56**:225-242 (July) 1941.

pilot's ears from external sounds. It is equally important to protect the ears of the members of the ground personnel and others exposed to noise of loud intensity.

Fowler [28] states in part in his summary of an excellent paper on tinnitus aurium:

> I have divided tinnitus into two fundamental types, vibratory and nonvibratory The first is mechanical in origin due to actual vibrations. The second is biochemical in origin and not due to any actual vibration . . . the loudness of tinnitus may be objectively estimated by comparing it to an applied tone or noise of known loudness.

In many instances, although the patient says that the noises in his head are so loud as to be unbearable, they are actually very faint, as determined by the loudness balance technic.

> The maskability of tinnitus by discrete and by band frequencies (thermal noise) is shown graphically with audiograms of different types of hearing loss. In some instances the tinnitus is very easily masked in all frequencies. In others the masking is greatest as the masking tone approaches near to the frequency band of the tinnitus. In others the masking is difficult, and in some it cannot be accomplished even by using the loudest masking sounds . . . nonvibratory tinnitus often sounds like escaping steam or air, or the sibilant "sh." Since this is exactly like the sound of a thermal noise covering all frequencies up to 10,000 in about equal intensity, it is suggested that hissing tinnitus is carried by the simultaneous irritation of a very large number of neural elements . . . neither the loudness of the tinnitus nor its frequency alone determines the ease of masking it. . . . The presence of tinnitus is always associated with more or less deafness . . . the frequency and loudness of tinnitus determine the deafness caused by the tinnitus lesions. This does not necessarily coincide with the deafness in general or the fluctuations of "speech hearing." The factor of fatigue from tinnitus or irritations causing tinnitus is discussed. The mechanism of the production of tinnitus is discussed in the light of neurophysiological research, and the absence of nonvibratory tinnitus in cochlear microphonics is pointed out . . . increases in the loudness of tinnitus are often attended by an elevation in tone and vice versa . . . it is impossible to demonstrate beats between nonvibratory tinnitus and applied pure tone, and only actual vibrations (not biochemical) can be heard by someone other than the subject. Biochemical irritations do not follow acoustic patterns, and this is one reason why nonvibratory tinnitus seldom if ever has the timbre of a pure tone. It is in fact only an "illusion of a tone" or an illusion of a combination of tones. It would appear that the difficulty in some instances in submerging or masking tinnitus cannot be explained wholly on the theory that there are insufficient or no sensory elements available in the peripheral apparatus to respond to the masking sound because the "recruitment phenomenon" would function as long as there were any available neural elements left, and this would make it possible to mask out the tinnitus, particularly if it were peripheral in origin. Tinnitus is an evidence of life and where there is tinnitus there is also present the "recruitment phenomenon." . . . The difficulty in masking may be due largely to a medial lesion or

28. Fowler, E. P.: Head Noises, Arch. Otolaryng. **32**:903-914 (Nov.) 1940; Tinnitus Aurium in the Light of Recent Research, Ann. Otol., Rhin. & Laryng. **50**:139-158 (March) 1941.

one central to the end organ. . . . If this is the true explanation, we have at hand a test for differentiating the types of nonvibratory tinnitus, peripheral, medial, and central. The mechanism of bilateral tinnitus, of peripheral and central origin, is discussed. Tinnitus, if sustained, is a definite sign of increasing or potential deafness. The correlation of all the observations with those in other fields of sensory perception is discussed, particularly the association of tinnitus with dips and troughs in the audiogram curve. Tinnitus is interpreted as representing an early irritation preliminary to subsequent degeneration. . . . Peripheral nonvibratory tinnitus is likened to a tickling, tingling, or itching on particular areas of the basilar membrane. Medial or central tinnitus represents a mass irritation of neural elements with little or no resemblance to the patterns obtained from an actual sound except a thermal noise.

VESTIBULAR PHYSIOLOGY

Many processes in the physiology of the semicircular canals are not fully understood. By ingenious experimental methods, Sand,[29] studying the semicircular canals of the fish, has reached some surprising and interesting conclusions. His summary is as follows:

1. The activity of the horizontal canals is confined to planes near the horizontal. The vertical canals are active in all planes, and work together in antagonistic pairs, but the pairs are different and specific for each of the three primary axes of rotation.

2. The tonic action of the semicircular canals is due to the spontaneous activity of hair-cells, which discharge a persistent rhythm of afferent impulses in the absence of external stimulation.

3. The two-way response of an individual semicircular canal, whose discharge is increased by rotation in one direction and inhibited by rotation in the opposite direction, provides a basis for the interpretation of the persistence of reflex responses to opposite sense of rotation in animals after unilateral extirpation of the labyrinth.

4. Excitation of the crista of the horizontal ampulla is evoked by ipsilateral acceleration and contralateral deceleration (and inhibition by contralateral acceleration and ipsilateral deceleration), but, owing to the physical properties of the cupula, the effects persist for about half a minute after acceleration or deceleration has given place to constant angular velocity or the stationary state, and thus a peripheral mechanism is provided which is adequate to account for the origin and duration of nystagmus and after-nystagmus.

Sand's description of the coupling of these canals in movements about the three principal axes is at variance with the work of Tait and McNally. The latter consider the utricle to be the synergist of the opposite vertical canal. Sand's researches can be correlated with the anatomic findings of Lorente de Nó, who has shown that each crista is supplied with nerve fibers of three different sizes. Both Poljak and McNally found that the nerve enters the crista in two portions, one on the utricular and one on the canalicular side. McNally divided each in

29. Sand, A.: The Mechanism of Acustico-Lateral Sense Organs in Fishes, with Special Reference to Problems in the Physiology of Semicircular Canals, J. Laryng. & Otol. **55**:449-457 (Oct.) 1940.

turn and found that a diminished efficiency of response occurred but that the one sense receptivity continued. Sand shows that there is a continuous discharge of action currents and that this discharge is increased by stimulation and diminished by inhibition. The counterpart to these reactions may perhaps be found in the three kinds of nerve fibers and their distribution to the end organ.

The waltzing guinea pig has been studied by Lurie.[30] This peculiar animal was first found by Professor Ibsen, of Kansas State College of Agriculture and Applied Science, Manhattan, Kan., in December 1929. The waltzing guinea pig not only tends to run in circles but also is deaf. The circling begins soon after birth and by the third or fifth day is pronounced. The animals do not respond to external sounds of any kind. The circling and deafness persist throughout the life of the animal, which inherits its deafness and waltzing trait in accordance with the mendelian law for recessive characteristics. The waltzing or circling is not caused by the vestibular sense organs, the utricle, the saccule or the semicircular canals. Instead, the waltzing is caused by a central nerve lesion which cannot be demonstrated by histologic study. Presumably the site of the lesion is the interpeduncular nuclei. The eyes show no nystagmus during circling; the drum membranes and middle ear structures are normal. Histologic examination of the vestibular end organs, vestibular nerve and vestibular nuclei shows that all these are normal. The deafness of the animals is due to degeneration of the organ of Corti. This degeneration occurs in a definite manner. First the external hair cells degenerate; later the internal hair cells go. Still later the cochlear nerve and the cells of the spiral ganglions degenerate. Degeneration in the stria vascularis cannot be correlated with changes found in the organ of Corti. According to Lurie, the macula of the saccule gives no evidence of being concerned with hearing.

Continuing the galvanic studies on guinea pigs, Buchanan and Ladd[31] sectioned the eighth nerve instead of removing the labyrinth. This was performed by drilling a hole through the skull directly over the internal auditory meatus. An insulated electrode was inserted into the internal auditory meatus and the nerve was destroyed by a 10 milliampere current. Fifteen days postoperatively the brains were sectioned, to make sure that the eighth nerve had completely degenerated. It was observed that the reactions to galvanic stimulation varied just as when the labyrinth was destroyed. Thus the authors found it impossible on the basis of such a test to differentiate disease of the labyrinth and disease of the eighth nerve. The fact that the galvanic falling reaction per-

30. Lurie, H. M.: The Waltzing (Circling) Guinea Pig, Ann. Otol., Rhin. & Laryng. **50**:113-129 (March) 1941.

31. Buchanan, A. R., and Ladd, L.: Galvanic Reaction in Animals, Arch. Otolaryng. **31**:113-120 (Jan.) 1940.

sisted after destruction of the eighth nerve indicates that some other mechanism must function in this reaction. What this mechanism may be is difficult to say. It may be that the tenth nerve, particularly through the division known as Arnold's nerve, which furnishes the sensory supply to the external auditory meatus, may transmit the stimulus to the proper brain centers, whatever they may be. Other nerves which must be considered include, of course, the fifth, the ninth, the tenth and the eleventh cranial nerve and possibly the upper cervical nerves. In an endeavor to determine the mechanism of the reactions, Buchanan [32] made lesions in the brain stem by means of a Horsley-Clarke stereotaxic instrument. Six animals had lesions in the lateral vestibular nucleus, 7 in the medial vestibular nucleus and 4 in the descending vestibular nucleus. As in the previous experiments, paradoxic reactions were increased on stimulation of the ears both on the injured and on the intact side. When the lesions were extensive, the number of paradoxic reactions from the intact side was about twice as great as that from the injured side. This was found to be true in animals with lesions in the labyrinth or in the eighth nerve. Significant increases in the number of milliamperes of current required to elicit galvanic falling reactions on stimulation of either ear were not observed, even in animals having extensive destruction of the vestibular nuclei or of the reticular formation. The galvanic current did not tend to be more effective when either the cathode or the anode was in contact with the ear. Caloric and postrotatory nystagmus could be elicited in these guinea pigs, although these responses were less marked than those noted in normal animals.

Buchanan [33] further reports on a study of 38 guinea pigs in which electrolytic lesions were produced in the brain stem in the region of the vestibular nuclei by the use of a Horsley-Clarke stereotaxic instrument. In 77 per cent there was a spontaneous deviation of the eyes. In all of the animals in which the electrolytic lesions were present in Deiters' nucleus, spontaneous deviation and nystagmus were present. Similarly, a deviation in the vertical plane was a significant finding when the lesion involved Dieters' nucleus. Thus, when the labyrinth and eighth nerve of a guinea pig are intact, a unilateral lesion in the brain stem which causes vertical spontaneous nystagmus probably involves Dieters' nucleus. Horizontal nystagmus was more commonly noted when the lesion involved the medial or descending vestibular nucleus. Perversion of induced nystagmus by caloric and turning tests appeared consistently enough to be of diagnostic significance.

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32. Buchanan, A. R.: The Galvanic Reaction in Guinea Pigs, Arch. Otolaryng. **31**:120-126 (Jan.) 1940.

33. Buchanan, A. R.: Nystagmus and Eye Deviations in Guinea Pigs with Lesions in the Brain Stem, Laryngoscope **50**:1002-1011 (Oct.) 1940.

The stimulation of the labyrinth by electric means has been resumed by Spiegel,[34] who performed experiments on cats. The labyrinth was stimulated by placing an electrode into the round window after the bulla ossea had been opened, or into a shallow groove chiseled into the promontory. This electrode was held rigidly in place. A current of 1 to 3 milliamperes was sufficient to produce a reflex reaction in the eyeballs, and stimulation of the facial nerve was avoided. A vacuum cup was placed over the cornea, with a thin needle sticking in the cup so that its shadow projected onto the recording paper of the string galvanometer. Eye jerks were elicited by stimulations up to a frequency of 27 per second. From stimulation at a higher rate, a tremor of 17 to 23 per second resulted. A frequency below 5 per second sometimes resulted in a rate of nystagmus higher than that of the stimulation, but a stimulation rate of 6 or more resulted in a corresponding nystagmus.

The reaction of the pupil to sound stimuli, to thermal stimuli and to rotatory and galvanic stimulation has been studied by Zanzucchi.[35] Experiments were conducted on pigeons both before and after the bony labyrinth was opened. Sound stimulation (tuning forks and Edelmann's whistle) caused nystagmic movements of the eye corresponding to the semicircular canal exposed and mydriasis accompanied with hippus, which persisted after ablation of the cochlea but disappeared after cocainization of the labyrinth. It was thus evident that these pupillary reactions were due to acoustic stimulation of the otolithic and ampullar structures. The use of physostigmine salicylate, atropine and morphine in large doses caused disappearance of the reactions, which, on the contrary, were not modified by instillation into the conjunctival sac of a 1 per cent solution of cocaine with an equal amount of a 1 : 10,000 solution of epinephrine bitartrate. Both hot and cold irrigation (with precautions to avoid cutaneous reflexes) through the auditory canal and directly to the uncovered semicircular canal caused mydriasis, usually preceded by a temporary miosis. The dilatation was not prevented by cocainization of the tympanic cavity but did not occur when the labyrinth was cocainized or removed. Chilling of the labyrinth caused disappearance of the pupillary reflex to sound; warming tended to accentuate this reflex. On stimulation by rotation the pupillary reactions consisted of contraction during the rotation, followed, when the turning was stopped, by a fugitive increase of the miosis and then by mydriasis and hippus. The phenomena were unchanged by the opening of a semicircular canal or cocainization of one or both labyrinths. The reaction was even accentuated by

34. Spiegel, E. A.: Rhythmic Stimulation of the Labyrinth, Arch. Otolaryng. **33**:572 (April) 1941.

35. Zanzucchi, G.: Experimental Study of the Relations Between the Pupil and the Labyrinth, Arch. ital. di otol. **52**:105 (March) 1940; abstracted, Arch. Otolaryng. **33**:320-321 (Feb.) 1941.

complete removal of the membranous labyrinth, made twenty to forty days prior to the test. With galvanic stimulation of the labyrinth the effect on the pupil was the production of mydriasis on closing and during the passage of the current, which was proportional to the intensity of the stimulus. The pupillary reactions persisted unchanged after anesthetization of the labyrinth. They became more intense immediately after ablation of the labyrinth and increased in intensitiy for several days; finally, as degeneration of the vestibular nerve and centers proceeded, the reactions became progressively less until they disappeared altogether. It is noteworthy that after the labyrinths were cocainized or removed, the reactions to sound and thermal stimuli disappeared, while those to rotatory and galvanic stimuli remained and were intensified. This behavior of the pupils recalls the paradoxic phenomena of Langendorff from resection of the cervical portion of the sympathetic trunk and the superior cervical ganglion. The paradoxic excitability of the pupil to rotatory stimulation after removal of the labyrinth led Zanzucchi to study the effect of reflexes of the neck on the pupil. His experiments revealed that rotation of the body (the head being fixed) and twisting of the neck produced miosis proportional to the amount of rotation; the reaction was not changed by removal of both labyrinths. The miosis, then, is a true pupillary neck reflex—a movement of the pupil associated with deviation of the eyes, itself a neck reflex. Since rotation produces a stretching of the muscles of the neck, the organism would react by contraction of the external muscles of the eye and of the sphincter of the pupil, the latter already rendered more excitable by the ablation of the labyrinth and the consequent degeneration of the nerve. This phenomenon may help to explain contradictory results of vestibular tests encountered in cases of disease of the ear and may permit a more precise localization of the lesion.

The sympathetic nerves of the ear have an effect on the eye quite unrelated to the vestibular apparatus. For in association with diseases of the middle ear following radical mastoidectomy, disturbances of the sympathetic nervous system result. Berberich [36] examined 27 patients and found unequal pupils in 9. In an additional 9 there were slight changes in the pupils, which were unmeasurable. This clinical observation confirms the knowledge of the sympathetic innervation of the middle ear. A stimulation of the sympathetic fibers on the medial tympanic wall is reflected in a difference in size of the pupils of the eyes. Therefore, in cases of pupillary disturbance it may be well to pay attention to the condition of the middle ear.

To test the vestibular portion of the labyrinth, nystagmus, past pointing and falling are elicited. Caloric, galvanic or rotatory tests are

36. Berberich, J.: Pupillary Reactions in Affections of the Ear, Laryngoscope **50**:555-558 (June) 1940.

usually employed. The caloric test is the easiest and the most commonly used. Hirsh [37] presents a new labyrinthine test, which he calls the waltzing test. The waltzing reaction results from stimulation of the horizontal semicircular canal. Five cubic centimeters of water at a temperature of 15 C. is used to irrigate the external canal of the ear. The subject stands with the head raised 60 degrees, to bring the ampulla of the semicircular canal above and the straight portion below. This is the optimum position for stimulation of the external semicircular canal. Both arms are stretched out straight. The eyes must be kept closed. The subject is then asked to flex and raise first one knee and then the other. In twenty to thirty seconds he begins to spin in the direction of the irrigated ear (i. e., the slow component of the nystagmus). In several seconds more the entire body begins to show a deviation, and a pronounced waltzing then occurs. The waltzing movement continues as long as stimulation of the labyrinth continues. Some subjects may make a complete revolution of 360 degrees, while others turn only 180 or 270 degrees. If the labyrinth is stimulated with a large amount of cold water, the subject will lose his balance and fall, thus failing to spin on his vertical axis. The waltzing reaction seems to come from the homolateral vestibular spinal tract. It is therefore possible, with the aid of this waltzing test, to make a topical diagnosis of a lesion in the posterior longitudinal bundle in the floor of the fourth ventricle. This test does not inconvenience the patient and the results can be recorded by photographs and can be observed more easily than nystagmus.

Simonton [38] and Grove [39] have thoroughly reviewed the literature on Ménière's symptom complex. Patients are suddenly afflicted with the well known triad of vertigo, deafness and tinnitus. The attacks tend to recur, but remissions are irregularly spaced and may last for years. Research as to the causation and treatment is therefore difficult.

Wright [40] presents some interesting figures in support of the theory that Ménière's disease occurs and that it is caused by infection. He properly objects to the term "focus of infection," which implies a single localized center of infection. He insists on a thorough search for all infective lesions. His surgical treatment consists of the removal of tonsils and teeth and the operative drainage of the sinuses. Of 84 patients with vertigo, 70 were cured and 4 others improved; of 68 deaf patients, the hearing of 70 per cent was improved; in 34 of 57 cases of

37. Hirsh, C.: The New Labyrinthine Reaction: "The Waltzing Test," Ann. Otol., Rhin. & Laryng. **49**:232-238 (March) 1940.

38. Simonton, K. M.: Ménière's Symptom Complex: A Review of the Literature, Ann. Otol., Rhin. & Laryng. **49**:80-94 (March) 1940.

39. Grove, W. E.: An Evaluation of the Ménière Syndrome, Ann. Otol., Rhin. & Laryng. **50**:55-70 (March) 1941.

40. Wright, A. J.: Further Clinical Observations on the Nature and Treatment of Ménière's Disease, J. Laryng. & Otol. **55**:388-393 (Aug.) 1940.

tinnitus there was improvement. These observations were made at least six months after surgical intervention. Success in 88 per cent of cases of vertigo is an extremely good record. In this country the removal of "foci of infection" is classed as one of the "general measures" used in the treatment of patients with Ménière's disease. The complete and careful eradication of all foci is of course essential and needs emphasis.

Grove [39] finds that the weight of evidence indicates that the main pathologic cause of Ménière's symptom complex is waterlogging, or edema, resulting from a metabolic disturbance. Whether this results from a disturbance of the water balance or from a disturbance of the sodium ion is not clear. In view of the fact that tinnitus persists after total destruction of the labyrinth, it may be that the waterlogging process, or edema, operates both in the end organ and in the central nuclei. All patients should have the opportunity of trying medical treatment. Dehydration, the elimination of sodium and treatment with histamine phosphate are measures which have been used with success.

Sheldon and Horton [41] reject the views which place pathologic changes associated with Ménière's syndrome in various cerebral structures and express the belief that the lesion is situated in the inner ear, that it consists essentially of localized alteration in the permeability of the capillary wall and that this causes regional edema. The labyrinth is held to be the specific site most frequently involved, because of the prominence of vertigo in the symptoms, and tinnitus and diminution of hearing are considered evidence of secondary cochlear involvement. In November 1939 these authors first treated a patient suffering from Ménière's disease by the intravenous administration of 1.9 mg. of histamine phosphate dissolved in 250 cc. of physiologic solution of sodium chloride. The time required for administering the solution is one and a half hours. This first patient, who had been confined to bed for three weeks with symptoms of Ménière's disease, was promptly relieved of dizziness and unsteadiness and was able to get up immediately after the injection was stopped and to walk normally. Fifteen additional patients responded to this form of therapy in the same spectacular manner.

Horton [42] believes that the perfect treatment for the disease has not been announced and will not be announced immediately. At the time of writing, 15 of 49 patients treated with histamine phosphate are known to be well. The others have had one or more recurrences of symptoms, possibly because they were not taking a maintenance dose. It should be

41. Sheldon, C. H., and Horton, B. T.: Treatment of Ménière's Disease with Histamine Administered Intravenously, Proc. Staff Meet., Mayo Clin. **15:** 17-21 (Jan. 10) 1940.

42. Horton, B. T.: Use of Histamine in Ménière's Disease, Surg., Gynec. & Obst. **72:**417-420 (Feb.) 1941.

remembered that while histamine phosphate is of undoubted value in the treatment of selected patients with Ménière's disease, it is a powerful vasodilator and that an excessive dose causes circulatory failure, owing to dilatation of the peripheral vascular bed. For this reason, intravenous administration of this drug should be very slow and cautious. It is safer to administer histamine phosphate subcutaneously.

Each exponent of the various types of medical treatment reports success with his method. Patients who have not been helped go to the neurosurgeon. Dandy [43] states that Ménière's disease can be cured permanently by division of the auditory nerve. This procedure carries almost no risk to life. Dandy has performed 401 operations, with 1 death, due to meningitis. In recent years, partial division of the nerve has been used when there is reason to save remaining hearing. The vestibular branch can be divided, leaving a part of the auditory branch intact. Usually, three fourths of the anterior part of the auditory nerve is sectioned, to insure total elimination of vestibular fibers. Division of even three fourths of the auditory branch of the nerve does not affect hearing (because of redundant fibers serving similar functions). If one auditory nerve—or only its vestibular division—is divided, there is no permanent loss of vestibular function of any kind. Immediately after operation the patient may or may not have transient, even severe, dizziness, not unlike an attack of Ménière's disease. This usually disappears in a few days. Dizziness on turning the head may persist for days, weeks or even months. This, too, gradually decreases and then disappears; only occasionally does it continue for a long time. Tinnitus may or may not disappear after section of the nerve; if it persists, it may or may not abate. In about 50 per cent of cases it disappears completely. There is no staggering, and the Romberg sign is negative after section of one nerve. Division of both vestibular nerves is attended by one rather surprising effect, visual jumbling of objects when the patient moves; when the patient is at rest, the objects are perfectly clear. Another disturbance is uncertainty when the patient is walking in the dark. Both of these persist, though with gradual decrease in severity.

43. Dandy, W. E.: Surgical Treatment of Ménière's Disease, Surg., Gynec. & Obst. **72**:421-425 (Feb., no. 2A) 1941.

Abstracts from Current Literature

Ear

FURTHER CLINICAL OBSERVATION ON THE NATURE AND TREATMENT OF MÉNIÈRE'S DISEASE. A. J. WRIGHT, Proc. Roy. Soc. Med. **33**:459 (June) 1940.

In substantiation of his concept that Ménière's disease is due to a focus of infection, chiefly in the nose, mouth or throat, Wright reports the results of treatment by removal of the focal infection in a large number of patients with the symptoms of this disease. The treatment consisted in the removal of infected tonsils, the removal of infected teeth and the operative drainage of infected accessory nasal sinuses. Of 84 patients having the symptom of vertigo 88 per cent were cured or improved; of 68 patients having the symptom of deafness 70 per cent were cured or improved, and of 57 patients having the symptom of tinnitus 59 per cent were cured or improved. Wright mentions cases from the literature and from his own experience which, with his convincing arguments, tend to substantiate his contention of the importance of infection as an etiologic factor in Ménière's disease.

CAMPBELL, Philadelphia.

DIAGNOSIS OF MASTOIDITIS IN THE INFANT BY PUNCTURE OF THE ANTRUM. H. GRENET, LALLEMANT, L'HIRONDEL and BOELLE, Bull. Soc. pédiat. de Paris **37**:438 (July) 1939.

A needle and a trocar, with the guard raised to prevent its entering too far, are inserted into the mastoid antrum as a diagnostic aid in cases of infants suspected of having mastoiditis. An antrum found to contain air which circulates freely through it is considered normal. If mucopurulent secretion is present a diagnosis of mastoiditis is made and treatment instituted. A few drops of solution of sodium chloride may be injected, aspirated and examined to determine whether pus is present. No untoward effects were encountered. The lateral sinus was punctured on one occasion, but no unfavorable result ensued.

BENJAMIN, Montreal, Canada. [AM. J. DIS. CHILD.]

ASEPTIC MENINGITIS RESEMBLING PURULENT MENINGITIS AND FACIAL PARALYSIS FOLLOWING ACUTE SUPPURATIVE OTITIS MEDIA. P. NOBÉCOURT and E. ROSENKOVITCH, Bull. Soc. pédiat. de Paris **37**:458 (Jan.) 1940.

A 7½ year old boy had acute suppurative otitis media. After myringotomy there was a purulent discharge for a few days. Facial palsy and purulent meningitis developed. No bacteria were demonstrated in the spinal fluid. He was treated with sulfanilamide. The pathogenesis of the meningitis is discussed, and the opinion is expressed that the disease was probably caused by a neurotropic virus and was not related to the middle ear infection. Sulfanilamide is given no credit for the boy's recovery because this type of meningitis usually clears up spontaneously.

BENJAMIN, Montreal, Canada. [AM. J. DIS. CHILD.]

GUSTATORY SENSIBILITY OF THE ANTERIOR TWO THIRDS OF THE TONGUE IN PATIENTS WHO HAVE UNDERGONE RADICAL MASTOIDECTOMY. G. TORRINI, Arch. ital. di otol. **52**:516 (Oct.) 1940.

No doubt exists that the gustatory sensibility of the posterior third of the tongue is supplied by the glossopharyngeal nerve, but there are still differences of opinion with regard to the innervation of the anterior two thirds. Torrini tested the taste

sense in the anterior two thirds of the tongue in 20 patients who had undergone a radical mastoid operation two months previously. On 7 of the patients the test was repeated after the lapse of three years. The test of Zwademacker, modified by applying the test solution with a medicine dropper, was chosen. Solutions of salt, sugar, tartaric acid and quinine in increasing strengths were applied to corresponding areas on the two sides of the tongue, and between each change of concentration the mouth was thoroughly rinsed with water; between tests with different substances the patient rested fifteen minutes. The initial test was made with the weakest concentration, and if no perception was noted, the next higher concentration was applied. The responses from the two sides were then compared.

In 2 patients complete ageusia was observed; in 3 others there was partial ageusia (for only some savors), but in most cases more or less marked hypogeusia on the side corresponding to that of the mastoidectomy was noted. In the last group it was observed that various parts of the tongue homolateral to the side of operation presented zones of complete ageusia. The results of the investigation confirm the anatomic fact that fibers which compose the lingual nerve cross the raphe and supply parts of the opposite side of the tongue. In addition, the results suggest that part of the gustatory fibers of the lingual nerve enters the chorda typani nerve and another part takes a direct course across the mandibular nerve, the otic ganglion and the third division of the trigeminal nerve. The distribution of the gustatory fibers probably differs in different persons, which would explain the diversity of responses in patients who have all suffered the same mutilation of the chorda tympani. Results with the patients on whom the tests were made three years after the operation show that no restitution of function had taken place on the side corresponding to the mastoid exenteration.

DENNIS, San Diego, Calif.

NOMENCLATURE OF THE VERTICAL SEMICIRCULAR CANALS AND RESULTANT ERRORS. G. E. JANNULIS, Monatschr. f. Ohrenh. **74**:353 (July) 1940.

To avoid error the term superior and posterior semicircular canal is advocated by Jannulis.

LEDERER, Chicago.

VESTIBULAR EXAMINATION OF PATIENTS WITH EXOPHTHALMIC GOITER. B. SZENDA, Monatschr. f. Ohrenh. **74**:557 (Nov.) 1940.

A series of 34 patients with exophthalmic goiter were examined otologically. The only complaints noted by the patients had been itching in the external auditory canal and slight tinnitus, all of which disappeared after surgical removal of the thyroid gland. The cochlear nerve was found to be normal, while the vestibular reaction to caloric stimulation showed a decrease in response in 25 to 50 per cent of the patients. With the decrease in the basal metabolic rate after operation the vestibular reaction to caloric stimulation returned to normal. The author believes that this unexpected reaction might be caused by the presence of sympathetic fibers in the vestibular ganglion. These fibers give a paradoxic reaction when they are permanently in a state of hyperirritability, owing to the increase of thyroxine associated with hyperthyroidism. Biochemically, this hyperirritability may be due to the increased oxidation, which disturbs the ion equilibrium of the endolymph and thereby changes the irritability of nerve endings. These findings may also be of practical importance, inasmuch as a very great number of patients suffering from Ménière's syndrome show signs of a hormonal disturbance, especially of the thyroid and generative glands. In patients with Ménière's syndromes with an increased basal metabolic rate a decreased vestibular reaction was observed, while the cochlear nerve was still normal. The caloric reaction returned to normal after reestablishment of a normal basal metabolic rate. Two case histories are presented.

LEDERER, Chicago.

LAW OF SYMMETRIC FUNCTIONAL LOSS OF HEARING IN INHERITED DEAFNESS. B. LANGENBECK, Monatschr. f. Ohrenh. **74**:562 (Nov.) 1940.

In the material at the ear, nose and throat clinic at Leipzig were included 297 cases of inherited deafness. When no later disturbance can be traced the following observation tends to support the rule of symmetric loss of hearing, as established by the author, namely, that in the majority of cases of inherited deafness there is a symmetric loss of function (more than 90 per cent). In about 10 per cent there are an unstable anlage and thereby asymmetric loss of function. Absolute proof of inherited deafness is found when two or more members of a family show symmetric hearing islands. Complete loss of function of the cochlea with preserved vestibular function has to be judged with care, inasmuch as such a finding may also be accounted for as the result of acquired deafness (1.5 per cent). Complete loss of vestibular and cochlear function does not tend to support this rule, as it is found only in 5 per cent of the cases of inherited deafness.

LEDERER, Chicago.

CARCINOMA OF THE EXTERNAL AUDITORY CANAL. MASAMORI MIYAZAKI, Oto-rhino-laryng. **13**:764 (Nov.) 1940.

Carcinoma of the external auditory canal is rare. Miyazaki collected 8 cases from the Japanese literature and added 1 of his own from the University Hospital, Soule, Chosen. The patient was a fisherman aged 58. His chief complaint was impaired hearing and a sensation of fulness on the left side. He gave no history of any previous aural disorder. About three years previous to coming to the hospital, he experienced intermittent itchiness about the external canal and used the stump of matches to scratch the ear; impaired hearing, tinnitus and fulness on the affected side gradually developed. Six months later he discovered mucoid otorrhea and consulted an otologist, who recognized a growth, the nature of which was not known. He was advised to have it removed surgically, but because he had no pain he refused operation.

Otoscopic examination showed that the growth entirely filled the external auditory canal. The tumor was grayish white, smooth on the surface and round and was attached to the lower portion of the wall of the canal. On probing it felt hard. There was no tendency to bleeding. There was no adenopathy, and no other abnormality was noted in the otolaryngologic examination. The Wassermann reaction of the blood was negative.

With local anesthesia the entire growth was removed in one piece. The bony wall was normal. The drum membrane was intact. After the operation the sensation of fulness and impairment of hearing disappeared. The wound healed in eighteen days. Histologic section of the tumor showed it to be an adenocarcinoma.

HARA, Los Angeles.

Pharynx

X-RAY TREATMENT IN INFECTIONS OF TONSILS AND PHARYNX. G. GHOSH, J. Indian M. A. **9**:413 (June) 1940.

The benefits derived from roentgen treatment of infected tonsils are sufficiently definite to warrant its use. The types of patients most suitable are (1) patients with frequent colds but otherwise good health; (2) patients with heart disease in whom the tonsils have been removed but infected tissue remains, and (3) patients with abundant lymphoid tissue in the pharynx not amenable to surgical removal.

TENNEY, Madison, Wis. [AM. J. DIS. CHILD.]

TONSILS AND ADENOIDS. LANCELOT BARRINGTON-WARD, Practitioner **145**:309 (Nov.) 1940.

The author concludes as follows:

"To sum up, it may be stated that the pathological effects of the tonsils are neither so black nor so white as they have been painted. Absolute indications for

tonsillectomy cannot be laid down, and the evidence requires careful sifting before judgement is given. In properly selected cases its performance confers a lasting benefit on the individual." GELSTON, San Francisco. [AM. J. DIS. CHILD.]

ACTION OF ROENTGEN IRRADIATIONS ON THE PHARYNGEAL TONSIL. F. CAVAZZANI, Arch. ital. di otol. **52**:591 (Dec.) 1940.

Cavazzani studied the effects of roentgen irradiation on the pharyngeal tonsil of 15 children, varying in age from 3 to 10 years. Four applications were made at intervals of one week, and the same technic was used for all. The majority of the children had clinical evidences of the presence of adenoids, and half of them had a family taint of tuberculosis. They were reexamined at intervals during periods of from one to twenty months after treatment. In 10 instances the pharyngeal tonsil was eventually removed surgically, and the present report concerns the histologic changes observed in the specimens removed. These changes are summed up thus:

1. Within one to four months after irradiation, modifications (areolar necrosis) of the lymphocytoblastic matrix, associated with slowing of the production of mature lymphocytes, attenuation of the lymphocytic halo, dissociation and fusion of the follicles and interfollicular, perivascular and pericanalicular fibroblastic proliferation were observed. 2. After this phase signs of replacement of the lymphoid elements by reticuloendothelial tissue appeared, and eventually (twenty months later) the adenoid follicles were reduced in number and volume and there were marked signs of sclerosis. DENNIS, San Diego, Calif.

FATAL SKI STICK INJURY OF THE SOFT PALATE. F. PRIETZEL, Monatschr. f. Ohrenh. **74**:309 (June) 1940.

A ski rod, pushed into the mouth of a youth while skiing, caused a slight laceration of the right tonsillar fossa and the soft palate. The next day there were signs of increased intracranial pressure, and death ensued in spite of a decompression. Autopsy revealed a traumatic lesion of the right internal carotid artery prior to its entrance into the skull, at the end of a wound canal from the soft palate, and an embolus in the right arteria cerebri media dextra. No bone injury was evident. LEDERER, Chicago.

POSTOPERATIVE ANGINA. HANS GERLACH, Ztschr. f. Hals-, Nasen- u. Ohrenh. **45**:428 (June) 1940.

Gerlach analyzed a series of cases of sore throat following septal operations, operations on sinuses and turbinates, removal of nasal polyps, the use of nasal tampons and nasal plastic operations. He concluded that sore throat was most common after septal operations. He considers it advisable to give the patient a course of treatment with the original prontosil (a hydrochloride of 4-sulfamido-2′, 4′-diaminoazobenzene) therapy prior to any intranasal operation. He states that since he has instituted the prophylactic administration of prontosil not a single case of septic sore throat following intranasal surgical treatment has been reported in his hospital. PERSKY, Philadelphia.

Larynx

ANTEROPOSTERIOR ROENTGENOGRAMS OF THE LARYNX. L. CIURLO and A. OLIVERI, Arch. ital. di otol. **52**:491 (Oct.) 1940.

Continuing their studies with roentgenograms obtained with the film in the hypopharynx, Ciurlo and Oliveri report further experience with the method. The present study concerns observations on 15 patients and is embellished with photographs and pertinent case histories. The patients suffered mostly from neoplasms and inflammatory and infiltrative conditions. It was clearly demonstrated that

anteroposterior roentgenograms can faithfully reproduce the image of the lesion discovered by laryngoscopy and in some cases they reveal elements which were not observed in the clinical investigation. This is particularly true in cases in which a satisfactory view of the larynx is not obtainable. Lateral and oblique roentgenograms furnish valuable information also and are not to be superseded by the anteroposterior projection, which is the indispensable complement of them. As to the objection that the introduction of the film is difficult, the authors state that they have never been obliged to resort to special methods of anesthesia and that they have never had to complain of annoying incidents.

DENNIS, San Diego, Calif.

SUBCUTANEOUS HEMATOMA OF THE NECK FOLLOWING VOCAL STRAIN. G. E. ARNOLD, Monatschr. f. Ohrenh. **74**:273 (June) 1940.

A man 20 years of age noticed a swelling on the left side of the neck just above the middle third of the sternocleidomastoid muscle. The swelling appeared suddenly after very loud singing. The diagnosis of a hematoma produced by the rupture of a superficial vein was made. Straining of an unaccustomed voice while singing was given by the author as the etiologic factor. A muscle herniation was first considered, but the clinical findings and the progress in the case were against this diagnosis. There was no fever. The author recorded kymograms of the speaking and the singing voice of the patient.

LEDERER, Chicago.

EXTERNAL LARYNGOCELE, WITH OPERATION. W. MEYER, Monatschr. f. Ohrenh. **74**:501 (Oct.) 1940.

Laryngocele may be either symptomatic, caused by obstruction to expiration, or congenital. It may penetrate through the hyothyroid membrane (external) or into the false cords (internal). Sometimes the two types are combined. According to an extensive roentgenographic survey, the lesion exists in about 5 per cent of all Europeans. A 37 year old man noticed a swelling on the right side of his neck. Dyspnea and dysphagia were not present. The swelling disappeared with a slight whistling noise under pressure from the surface and refilled under conditions of cough. A roentgenogram confirmed the diagnosis of laryngocele. Excision was performed, and the pedicle of the sac could be traced to its permeation through the hyothyroid membrane. Histologic examination of the sac revealed a single layer of ciliated columnar epithelium supported by loose connective tissue.

LEDERER, Chicago.

PATHOGENESIS OF CONGENITAL INSPIRATORY STRIDOR. FRANZ ANGYAL, Pract. oto-rhino-laryng. **3**:129 (Aug.) 1940.

The author describes a case of congenital inspiratory stridor in a child 2½ years old, who subsequently died of bronchopneumonia. Autopsy revealed marked folding of the epiglottis and enlargement of the aryepiglottic folds. These seemed to form flaps and so caused marked narrowing of the aditus of the larynx. There was complete absence of musculature in the aryepiglottic fold. In his opinion, there was a congenital alteration of the larynx with exaggeration of the infantile type, and the absence of the muscle in the aryepiglottic fold was definitely the pathologic basis for the condition.

PERSKY, Philadelphia.

Nose

COMPLICATIONS INCIDENT TO PARANASAL SINUSITIS. FRANCIS W. WHITE, Pennsylvania M. J. **44**:141 (Nov.) 1940.

In this paper the author does not discuss diagnosis or the ordinary treatment of paranasal sinusitis. The basis of study is a series of 103 patients treated in

the Manhattan Eye, Ear and Throat Hospital over a five year period, who had or later acquired complications. The group represents 5 per cent of all patients hospitalized for treatment of sinusitis. The average period of hospitalization was twenty-two days. Orbital complications were the most common. A working syllabus for diagnosis and treatment in use in the clinic is cited as follows: (1) inflammatory edema of the eyelids, with or without extension of edema into the orbit; (2) subperiosteal abscess, with (*a*) edema of the lids and orbit or (*b*) spread of the pus to the lids (erroneously called orbital abscess); (3) orbital abscess; (4) orbital cellulitis, either (*a*) severe or (*b*) mild, and (5) thrombosis of the cavernous sinus.

The author points out that in osteomyelitis edema is the first and most important of all signs and precedes the roentgenogram in diagnostic importance by seven to ten days or more, owing to the slow loss of calcium in the infected bone. A correct diagnosis was made in 7 of 11 cases before the roentgenogram demonstrated its presence.

The mortality in the author's series of 11 cases of osteomyelitis was slightly over 27 per cent, and all of the deaths were the result of the postoperative type of osteomyelitis. Radical operation, up to removal of the whole calcarium in some cases, is advised. The author believes that the radical treatment will in time be so modified that hospitalization of the patients will be greatly shortened.

WEINBERGER, Sayre, Pa.

RELATION BETWEEN ACUTE PNEUMONOPATHY AND THE PARANASAL SINUSES. F. GREGORINI and G. SAIBENE, Arch. ital. di otol. **52**:555 (Nov.) 1940.

Gregorini and Saibene made histologic studies of the mucosa of the paranasal sinuses removed from 36 cadavers of persons who had died of pneumonia. The ages of the subjects varied from 16 to 87 years. In 17 of the 36 cases acute croupal pneumonia occurred and in 14 acute bronchopneumonia; in 5 foci of bronchopneumonia were added to the frank lobar type. The results of the examination are classified in two tables, one containing the macroscopic and the other the microscopic observations. The article is enriched with photomicrographs.

Congenital anomalies of the sinuses were observed in 5 cases: unilateral aplasia of the frontal and the sphenoid sinus, respectively, in 1 case each; marked hypoplasia of the sphenoidal sinuses in 2 cases, in 1 of which there was a single, medially situated frontal sinus without septums and in the other a single central frontal sinus with indications of septums. In few cases was there evidence of mucopurulent exudate. Polypoid vegetation was not seen in any case, and in only 3 cases was there cystic degeneration. The changes microscopically encountered were in general of the chronic type, subacute lesions being revealed in only 4 cases. In 8 of the 17 cases of pulmonitis signs of inflammation in the sinuses were observed, while such signs were present in only 2 of the 14 cases of bronchopneumonia and in 3 of the 5 cases of associated pneumonia. The epithelial layer was well conserved as a rule; sometimes thickening due to marked development and more rapid differentiation of the basal cells and sometimes numerous goblet cells were present.

The mucosa of the paranasal sinuses is subject to great individual variation, attributable frequently to constitutional factors and also to simple aging of the tissues. If sinusitis has real importance as a forerunner of pneumonia, it is difficult to explain the rare and modest histologic alterations observed in the sinuses. "The results of our research do not permit us to confirm surely the supposed importance of inflammation of the paranasal cavities in the pathogenesis of acute pulmonitis and bronchopulmonitis."

DENNIS, San Diego, Calif.

DIAGNOSIS AND THERAPY OF INFLAMMATION OF THE MAXILLARY SINUS. G. CLAUS, Monatschr. f. Ohrenh. **74**:287 (June) 1940.

Of 531 cases of operation on the maxillary sinus, the origin was nasal in 67.2 per cent and dental in 32.2 per cent. To investigate the thickness of the mucosa,

the sinus should be washed and then 0.5 to 1 cc. of fluid injected into the cavity through the irrigation needle, the head being tilted backward. Backflow of the fluid indicates thickening. A 10 per cent solution of 10 per cent iodoform and glycerin is advised as the therapeutic agent for irrigation, and it has the advantage of being radiopaque for roentgen studies.

LEDERER, Chicago.

OSTEOMA OF FRONTAL SINUS: REPORT OF A CASE, WITH OPERATION. T. BAJKAY, Monatschr. f. Ohrenh. **74**:293 (June) 1940.

An osteoma, the size of a pea, was removed from the right frontal sinus by trephine. No complication followed. The well known clinical course and pathologic features of such osteomas are discussed.

LEDERER, Chicago.

SHORT WAVE THERAPY OF ACUTE PURULENT SINUSITIS. F. PRIETZEL, Monatschr. f. Ohrenh. **74**:496 (July) 1940.

A series of 64 patients with acute suppurative sinusitis were given short wave therapy and the results compared with those for a similar number of patients treated in accordance with the usual method of shrinkage and application of dry heat bath to the head. An average of seven treatments of about ten to fifteen minutes each, with a voltage of 17 to 19 volts, was employed. In the average case restitutio ad integrum was achieved five days earlier than with the usual method. No operation was required, and the short wave treatment was found to be agreeable to the patient.

LEDERER, Chicago.

HARD PAPILLOMA OF THE NOSE. PAUL KEPES, Pract. oto-rhino-laryng. **3**:157 (Aug.) 1940.

The author, after presenting a case of a hard papilloma of the nasal cavity occurring in a man aged 42, which involved the entire right side of the nose, the right antrum and the right ethmoid sinus, compared observations in this case and those in similar cases reported in the literature. He believes that, while usually benign, the tumor may undergo malignant degeneration. Carcinomatous changes, with metaplastic islands occurring in the mucous membrane and deeper tissue, may be present. The cause is not clear.

The author recommends complete extirpation of the tumor, either intranasally or through a radical maxillary approach according to Denker's technic. Should extensive bleeding occur it may be necessary to ligate the external carotid artery. He also supplements the surgical procedure with high voltage therapy.

PERSKY, Philadelphia.

Miscellaneous

THE EFFECT OF NICOTINIC ACID ON BLOOD COAGULATION. R. M. CALDER and G. P. KERBY, Am. J. M. Sc. **200**:590 (Nov.) 1940.

Clinical experience of the authors in a large group of patients with chronic brucellosis, which they had previously shown to be associated with delayed clotting time, revealed that nicotinic acid possesses the property of promoting coagulation of the blood. In these patients subcutaneous hemorrhages often developed after slight trauma and the clotting time was slow, with imperfect retraction of the coagulum and low yield of serum. Administration of nicotinic acid remedied these defects. Clinical trial of nicotinic acid in treatment of other hemorrhagic states with delayed clotting time which was apparently on a toxic, infectious basis gave encouraging results. The dose was 100 mg. three times daily, given by mouth. A 0.6 per cent solution of nicotinic acid applied to the tooth sockets after extraction caused a firm, permanent clot in one to two minutes, whereas untreated sockets in the same patient often bled for several hours.

Experimentally it was found that nicotinic acid could not be substituted for any of the factors involved in normal coagulation but that when added in vitro to blood containing excessive amounts of antithrombin, such as heparinized blood or blood from dogs in peptone or anaphylactic shock, nicotinic acid does induce coagulation. That this effect was not due to nonspecific alteration of the p_H was shown by control testing with seven other acids. The authors believe, however, that since the doses used clinically are so small as compared with the enormous doses used in the in vitro experiments it is not justifiable to assume that the action of the nicotinic acid in patients is a chemical neutralization of antithrombin, though this may be possible. The nicotinic acid may correct a fundamental deficiency in the organism or induce the formation either of more prothrombin or of less antithrombin. The authors believe that further trial of nicotinic acid in treatment of various hemorrhagic states is warranted.

SHAMBAUGH JR. and JENNES, Chicago.

ORAL VS. PARENTERAL POLLEN THERAPY: A CLINICAL STUDY. BERNARD P. ALPERSTEIN, J. Allergy **11**:498 (July) 1940.

This is a chronologic review of the subject of pollen therapy, which reveals no unanimity of method, no standardization of dosage and no control for comparison. The purpose of this study was to evaluate the clinical, or therapeutic, effects of the oral method of giving pollen therapy as compared with those of the parenteral mode of administration. The results for a control group are included. The author's conclusions are:

"1. The oral method of pollen therapy gave satisfactory clinical relief in ragweed-sensitive patients who had not been treated in prior seasons; those who had had the parenteral form of therapy in previous seasons benefited much more from the oral form than those who had never been treated before.

"2. Neither the site of allergic manifestations nor the degree of sensitivity bore any relation to clinical relief of symptoms under any mode of treatment.

"3. Unpredictable reactions occurred with the oral method. The range of dosage (by the oral method) within which reactions occurred was quite wide, anywhere from 1,000 to 90,000 Noon units. The reactions were of three types, gastrointestinal, respiratory, and systemic.

"4. The parenteral method gave a greater percentage of satisfactory relief as well as a smaller percentage of complete failures than did the oral method.

"5. In view of the unpredictable reactions, a rigid schedule for oral pollen therapy cannot be planned for self-administration by patients. It is thus inadvisable to allow patients to treat themselves by the oral method.

"6. The plan of therapy for the subcutaneous administration of pollen extracts is amenable to individual needs and flexible enough to serve as a guide to obtain optimum results with maximum dosage.

"7. The experiments with the blood and urine of patients treated orally show that (a) an appreciable amount of pollen is absorbed through the gastrointestinal tract; and (b) that reagin and allergin coexist in the circulating blood at the same time at the height of treatment.

"8. The oral method is not recommended inasmuch as it has no particular advantage over the parenteral method; nor do the results obtained (by the oral method) show nearly as much beneficial clinical relief as those obtained by the parenteral method."

HOYER, Cincinnati. [AM. J. DIS. CHILD.]

MY TRANSSEPTAL OPERATION ON THE TEAR SAC. K. KOFLER, Monatschr. f. Ohrenh. **74**:603 (Dec.) 1940.

The author describes his modification of the West-Polyak operation on the tear sac. He reports 100 per cent good results in a total of 220 such operations and attributes his success to three factors: (1) improvement of visualization and

accessibility of the field of operation through a septal incision, (2) use of all experience gained from previous failures and (3) precise and radical surgical technic. Indications and contraindications are discussed. It may be noted that old age, ozena and even chronic suppurative sinusitis are not contraindications to the operation. The instruments necessary for Kofler's technic are described and illustrated. Anesthesia may be general or local. Local anesthetization of the septum is produced in the customary way, while the tear sac is anesthetized by injecting the solution around it from the outside (crista lacrimalis) and into the nasal mucous membrane. The operation consists of two parts, the procedure involving the septum and that involving the tear sac. The first incision into the septal membrane should be made close to the limen nasi in a straight line on the opposite side of the tear sac to be operated on. About 5 mm. deeper the cartilage is incised, or resected in cases of deformities, and the mucous membrane of the other side is cut at the same level. The specially constructed nasal speculum is now introduced into this septal incision, and its spreading gives an excellent approach to the region of the tear sac. Sometimes Hofer's incision, a straight incision through all septal layers, can be employed, especially in children, in whom the cartilage is not too hard to be pushed out of the way by the speculum. Both types of incision heal perfectly, even when they are repeated several times on the same person. With the aid of several illustrations, the different stages of the operation on the tear sac are described. The flap of mucous membrane must be formed properly, and complete exposure of the sac has to be achieved to the extent that not only the bony layer covering it but a larger bullar cell or an enlarged head of the middle turbinate bone is removed, to avoid secondary adhesions. A loose local tampon is allowed to remain in place for about a week, supported by septal tampons. Later, 5 per cent silver nitrate is applied topically. The great advantages of the transseptal approach are the better use of the chisel, which can attack the bone vertically, and the excellent inspection of the field. The possibility of exposing periorbital fat and the creation of nasal adhesions are the difficulties which may be encountered. A good result from the operation can be expected when a sufficient amount of the medial bony wall and tear sac wall has been removed in all directions without injury to the outer wall. A probe, introduced into the canaliculus, should be moved freely in all directions. The inner opening of the canaliculus should be about 5 mm. distant from all bony and membranous wall. Recurrence will be noted, if it occurs, after six to eight weeks, and reoperation has to be carried out according to the complications.

LEDERER, Chicago.

AGRANULOCYTOSIS ASSOCIATED WITH CERTAIN CHANGES IN THE NASAL MUCOUS MEMBRANE: REPORT OF A CASE. A. HEINDL, Monatschr. f. Ohrenh. **74**:630 (Dec.) 1940.

The pathologic disposition of the bone marrow is held responsible for agranulocytosis. Thereby the resistance of the mucosa to secondary invaders is diminished, and extensive ulcerations and necroses are present in the tonsils, tongue, etc. Involvement of the nasal mucosa is rare. Chronic sinusitis was held as the causative factor in 2 cases of agranulocytosis with ulcerations in the nose, but in the case described in this paper influenza was supposedly the cause of the fatal disease. Besides the general changes typical of this type of blood dyscrasia, nasal examination made a few hours before death disclosed a certain anesthesia of the nasal mucosa, with pallor and a yellow serous exudate. Autopsy revealed the typical changes. Examination of a specimen of mucosa from the nose showed that the mucosa had undergone degenerative changes of varying intensity, more marked in the inferior portion of the nose, while the olfactory fissure remained unchanged. Edema, destruction of the epithelium and bacterial thrombi were present, predominating on the right side. The mucosa of the maxillary sinus remained unchanged; that of the ethmoid cells was edematous. There is no doubt that these changes had been caused by secondary invaders which had been activated by the diminished resistance of the patient.

LEDERER, Chicago.

Book Reviews

Cancer of the Face and Mouth. By Vilray P. Blair, M.D., Sherwood Moore, M.D., and Louis T. Byars, M.D., St. Louis. Price, $10. Pp. 599, with 260 figures and 64 plates. St. Louis: The C. V. Mosby Company, 1941.

This remarkable book is a clinical treatise based on the personal observation, made during the past twenty years, of growths about the face and mouth, by Dr. V. P. Blair, one of the world's outstanding maxillofacial surgeons, in collaboration with Dr. S. Moore, well known professor of radiology at the Washington University School of Medicine and director of the affiliated Edward Mallinckrodt Institute of Radiology.

Material based on over 1,500 cases has been arranged in a regional manner, so that a physician seeking information on a definite problem can find it directly. Chapters on general surgical principles and general radiation therapy are followed by separate chapters on cancer of the following regions: the face, ear, external nose, orbit, lips, tongue and pharynx, cheek and jaws, nasal cavity and nasopharynx and the sinuses. Tumors of the skin and tumors of the salivary gland are also considered. Each chapter is profusely illustrated by a superb collection of photographs of lesions as they occur in patients. In many cases follow-up photographs made during treatment and photographs showing the result years later are included. There are over four hundred photographs, of highest quality. The follow-up photographs are a valuable aid in evaluating the result of therapy and of plastic surgical rehabilitation. For those interested in more detail as to specific management of cancer of the face and mouth there is a chapter on metastases in cervical nodes and neck dissection, a detailed chapter on end results secured in this type of work and a separate section of two hundred and sixteen pages on surgical technic. This section consists of descriptive matter and full page black and white sketches demonstrating the surgical procedure employed in the cases which are illustrated by photographs in the front part of the book. There is an excellent index.

The plan of the book is one of visual education and description based on personal observation and management. It will be of great value to students, to general practitioners in their daily diagnostic work and to medical specialists and pediatricians, general surgeons and the various specialists—dermatologists, dentists, oral surgeons, ophthalmologists, otolaryngologists, radiologists and plastic surgeons.

Pathology of the Oral Cavity. By Lester Richard Cahn, D.D.S. Price, $5.50. Pp. 240, with 165 illustrations. Baltimore: The Williams and Wilkins Company, 1941.

The author discusses the morbid processes of the more common oral lesions in an interesting manner and in a style that makes for easy reading. Much is compressed in two hundred and forty pages, and the value of the text is enhanced by the one hundred and sixty-five illustrations which clearly depict the subject matter under discussion.

There are in all nineteen chapters, dealing with the following topics: principles of inflammation, diseases of the hard structures of the teeth, diseases of the pulp, diseases of the periapical tissues, osteomyelitis, pyorrhea alveolaris, cysts of the jaw bones, other pathologic conditions of the jaw bones, tumors of the jaw bones, general histology and pathology of the oral soft tissues, pigmentation in the mouth, gingivitis and stomatitis, ulcers of the mouth, oral lesions of the soft tissue associated with blood dyscrasias, lesions of the oral soft tissue associated with endocrine dysfunction, lesions of the oral soft tissue associated with avitaminosis, pathologic conditions of the tongue, leukoplakia and tumors of the oral soft tissues.

There are a number of references at the end of each chapter, which the reader may consult if he wishes to study the subject more fully.

The index and the printing, binding and entire format of the text are first-class.

This textbook is recommended to all otolaryngologists seeking help in diagnosing lesions of the oral cavity, and to all doing oral surgical operations on the mouth.

Directory of Otolaryngologic Societies *

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Gordon F. Harkness, 215 Main St., Davenport, Iowa.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia.
Place: Atlantic City. Time: June 8-12, 1942.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Ralph I. Lloyd, 14–8th Ave., Brooklyn.
President-Elect: Dr. James A. Babbitt, 1912 Spruce St., Philadelphia.
Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: San Francisco. Time: Oct. 11-15, 1942.

American Broncho-Esophagological Association

President: Dr. W. Likely Simpson, 899 Madison Ave., Memphis, Tenn.
Secretary: Dr. Paul Holinger, 700 N. Michigan Ave., Chicago.
Place: Hotel Dennis, Atlantic City, N. J. Time: June 8-9, 1942.

American Laryngological Association

President: Dr. T. E. Carmody, 277-16th St., Denver, Colo.
Secretary: Dr. Charles J. Imperatori, 108 E. 38th St., New York.
Place: Atlantic City, N. J. Time: May 25-27, 1942.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. James A. Babbit, 1912 Spruce St., Philadelphia.
President-Elect: Dr. James G. Dwyer, 375 Park Ave., New York.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.
Place: Atlantic City, N. J. Time: June 1-3, 1942.

SECTIONS:

Eastern.—Chairman: Dr. Henry P. Johnson, 32 Deering St., Portland, Maine.

Southern.—Chairman: Dr. William L. McDougall, 915 Doctors' Bldg., Atlanta, Ga.

Middle.—Chairman: Dr. William E. Sauer, 3720 Washington Blvd., St. Louis, Mo.

Western—Chairman: Dr. Emil F. Tholen, 1136 W. 6th St., Los Angeles.

American Otological Society

President: Dr. E. M. Seydell, 107 W. Douglas Ave., Wichita, Kan.
Secretary: Dr. Isidore Friesner, 101 E. 73d St., New York.
Place: Atlantic City, N. J. Time: May 28-29, 1942.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.